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ASSOCIATION OF OSTEOMYELITIS OF THE SKULL AND NASAL ACCESSORY SINUS DISEASE

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This communication will discuss the cases of osteomyelitis of the skull which are found to complicate acute and chronic disease of the nasal accessory sinuses. Only cases due to infection by the ordinary pyogenic bacteria will be considered, and no case of infection by the tubercle bacillus, by the spirochete of syphilis or by Actinomyces will be included.

There will be no attempt made to discuss the essential nature of disease of the nasal accessory sinuses, as that is not my subject. Only that part of it which seems necessary to the proper explanation of the mechanism by which osteomyelitis of the skull bones complicates the accessory sinus disease will be included. In considering this subject it is to be remembered that not infrequently caries and necrosis of portions of the bony walls of the nasal accessory sinuses occur as part of the pathologic process incident to sinus disease itself; the bone involvement is strictly limited in extent to the area of the latter and directly underlies the affected portion of the mucous membrane. This is the so-called "sinusitis abscendens"; it shows but little tendency to spread beyond the limits indicated, and surgical intervention of a comparatively simple kind results in a cure. This type and variety of osteomyelitis is also not included in this communication.

Although osteomyelitis of the skull must have undoubtedly occurred frequently prior to the eighteenth century as a consequence of civil and military injuries about the head, the association of the injury and the subsequent clinical manifestations with infection of the bony tissue of the skull was not recognized until 1879, when von Bergmann and Lannelongue first pointed out that cranial para-osseous abscesses were due to disease of the bone itself. Luc and Tilley were the first to recognize the association of infection of the cranial bones with, and as a complication of, accessory nasal sinus disease, and each of them separately described cases before the British Medical Association in 1899. Up to the present time accurate scientific knowledge concerning the pathogenesis and mechanism of osteomyelitis of the skull complicating disease of the accessory nasal sinuses is mostly due to the work of Schilling, Ropke,

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Gerber and Luc. A résumé of the available literature of importance was made in 1913 by McKenzie, who collated the knowledge of the subject up to that date, the important contributions being from German and French sources. The original paper of Schilling contained the reports of 9 cases; Gerber added 20, and Luc increased the total number of reported observations to 39. McKenzie added 7 cases more.

Between 1924 and 1931, there were admitted to Mount Sinai Hospital, 326 patients with nasal accessory sinus disease; 12 instances were encountered in which some form of osteomyelitis complicated the picture. This is in accordance with the statement just made.

The cases in the Mount Sinai Hospital were distributed as follows:

Up to 10 years	3 cases
11 to 20 years	3 cases
21 to 30 years	1 case
31 to 40 years	1 case
41 to 50 years	2 cases
51 to 60 years	2 cases

Most of Burger's patients were between 11 and 30 years old. In Gerber's 29 cases, the age groups were as follows:

Up to 10 years	1 case
11 to 20 years	4 cases
21 to 30 years	11 cases
31 to 40 years	3 cases
41 to 50 years	3 cases
51 to 60 years	0 cases
Over 60 years	1 case

In the observations up to now, the female sex has preponderated. The ratio to the male sex is 8:1 according to Schilling, 11:5 according to Ropke, 16:9 according to Burger and 13:14 according to Gerber. Gerber does not believe that from these figures one may draw a conclusion regarding the preponderance of the female. At any rate, this seems to be of little importance.

There was an involvement of the right side in 5 cases, of the left side in 4, and of both sides in 10, among the cases reported by Ropke.

LOCATION OF THE ORIGINAL SINUSITIS

As regards the accessory sinus in which the infection originates, it must be remembered that in clinical practice there seems to be no strict limitation of the infected area to any regional group of sinuses; indeed the usual thing is to find a diffuse process involving all the accessory sinus groups. If, as sometimes happens, there seems to be a starting point for the infection at some particular area, the spread from the latter to the others is extremely rapid. It would be better to

say that the sinus infection, although multiple, seems to exhibit greater manifestations in one or the other groups of nasal accessory sinuses.

With these criteria in mind, it would, then, be proper to accept McKenzie's figures, namely, that osteomyelitis of the cranium occurred 45 times as a consequence of frontal sinus disease as opposed to 3 times as a consequence of antral disease in a total number of 48 cases.

McKenzie found but a single case originating in the ethmoid cells: a case of van den Wildenberg's. In this case a small sequestrum of the unguis was first removed, and fourteen days later a large sequestrum comprising the crista galli and the lamina cribrosa. From observations to be detailed subsequently, I am inclined to agree with van den Wildenberg and to disagree with McKenzie, who wished to classify this case also as having originated in the frontal sinus. The case of Sir St. Clair Thomson to which McKenzie made reference in this connection had better be omitted from the discussion at present, as the mechanism was complicated by the performance of an operation.

The experience of other surgeons varies. Thus Jansen in 10 instances, Ropke in 37, Killian in 16 and Luc in 4 found that the ethmoid cells were affected along with the frontal sinus. Walker-Downie noted ethmoid disease in 5 of 6 cases. On the other hand, Lermoyez, in 36 instances in which the frontal sinus was affected, found only 2 with associated ethmoid disease, while Herbert Tilley, in 25 diseased frontal sinuses noted 6 with ethmoid suppuration. Milligan described disease of the ethmoid cells in 18 of 40 cases, while of 20 diseased frontal sinuses observed by Turner the ethmoid cells were also affected in 12. In Wertheim's postmortem observations, the ethmoid cells were found to be affected in 7 of 12 cases. In a total of 206 instances in which chronic suppuration of the frontal sinus existed, the ethmoid cells were implicated in 117, i. e., in 56 per cent. It is difficult to assign a cause for the appreciable variance in the individual compilations of cases. One of the things to be remembered is that the differentiation of the areas that seem to bear the major part of the infection in the general range of the nasal accessory sinuses is always more or less dependent on the individual interpretation of the objective findings. Misinterpretation because of obscure localization of the focus is also possible. It is easily appreciated how this could account for the differences in the available statistics.

The antrum of Highmore[1] is a relatively rare precursor of an osteomyelitis of the skull. One finds few references to this etiologic connection. In a case of Haymann, the sinusitis in the antrum coexisted

1. The subject of osteomyelitis of the maxilla was discussed in another communication (Wilensky, A. O.: Osteomyelitis of the Jaws, Arch. Surg., to be published).

with an infection of the frontal sinus; it is much more likely that the osteomyelitis of the skull was related to the frontal sinusitis rather than to the infection of the antrum. And it is quite likely that a similar, and possibly a frequently unrecognized, condition in a frontal sinus was present in the reported cases of antral disease in which osteomyelitis of the skull resulted as a complication.

Among the 12 cases of osteomyelitis of the skull after disease of the nasal accessory sinuses in the records of the Mount Sinai Hospital the distribution of the original sinus disease was as follows: frontal, 6 cases; ethmoid, 2 cases; sphenoid, 3 cases; frontal and ethmoid, 1 case, and sphenoid and ethmoid, 1 case. I was unable to find any case of osteomyelitis of the skull after disease of the antrum.

VARIETIES OF SINUSITIS

Osteomyelitis of the skull follows both the acute and the chronic variety of nasal accessory sinusitis. It is altogether likely that even in cases of chronic sinusitis, the occurrence of the bone complication is always in association with an acute flare-up of infection and a spread of the latter into new areas. The only exceptions to the last part of this statement are found in the simple perforations, to be described.

BACTERIOLOGY

The bacteriology of osteomyelitis of the skull under the conditions discussed in this communication must necessarily be somewhat uncertain, as bacteriologic examination must frequently show contamination with other organisms the habitat of which, under normal conditions, is the nose and the nasal accessory sinuses. The usual bacteria found as described in the literature include Staphylococcus aureus, streptococci and pneumococci. The statement is made by Skillern in discussing Woodward's paper that Staphylococcus aureus cases all progress well and that the streptococcus and pneumococcus cases are all fatal. The statement is apparently not sufficiently supported by clinical and bacteriologic evidence and needs considerable clinical confirmation before it is accepted.

VARIETIES OF OSTEOMYELITIC BONE LESIONS FOUND

In the literature the varieties of osteomyelitis of the skull that complicate nasal accessory sinus disease have been differentiated and classified on the basis of the extent of the lesion and the rapidity of spread of the pathologic process. This form of classification seems to be a good one and will be followed in this communication also. Clinically the following are found:

1. The osteomyelitis takes the physical form of a perforation. In a certain number of the cases this is associated with insufficient, or with

absence of, drainage from the normal ducts, and the character of the pathologic changes leads one to believe that the process is just what its name implies: the perforation of an abscess through a bony wall. It is questionable whether this form ought to be classified as an osteomyelitis; personally, I do not think so.

Perforation is quite common, however, with well marked and more widely spread evidence of true inflammation of bone and of necrosis and sequestrum formation. In these, the diagnosis of osteomyelitis is never in doubt, and the perforation—in contradistinction to the type just described, in which it is just an attempt on nature's part to secure drainage—is a matter of little importance in the total clinical and pathologic picture.

Perforations are found to complicate both the acute and the chronic type of accessory sinus disease.

A. In the cases of frontal sinus disease the perforations occur as follows:

(*a*) In the anterior wall of the frontal sinus. An abscess then forms over the frontal eminence. The spread of this form of abscess is always upward under the scalp.

(*b*) In the floor of the frontal sinus. A subperiosteal abscess forms which can be palpated as a lump attached to the roof of the orbit. Involvement of the general orbital cavity cannot and does not occur until the orbital periosteum breaks and permits the diffusion of the pus into the orbital cavity. In the late stages proptosis occurs. This form of abscess never spreads upward under the scalp.

(*c*) In the roof of the frontal sinus. An extradural abscess forms. The further spread of the suppuration occurs by tracking between the dura and the bone and by increasing accumulations of pus, when signs of intracranial compression occur; or by perforation through the dura when subdural intracranial complications, to be discussed subsequently, occur.

B. In the cases of ethmoid disease perforations occur as follows:

(*a*) Into the orbit. An orbital phlegmon results.

(*b*) Into the nasal cavity. This is usually accompanied by an amelioration of the symptoms and is otherwise unnoticed in the clinical picture.

C. In the cases of sphenoid disease perforation occurs as follows:

(*a*) Through the floor or anterior wall of the sphenoid and into the superior pharynx. Cases are reported in which a tumefaction is palpated in the roof of the pharynx which, when opened, is found to be referable to a perforation in the appropriate wall of the sphenoid. Undoubtedly this happens more often clinically and is unrecognized

owing to the fusion of the discharge with that from the natural opening or from adjacent areas.

(*b*) Through the roof of the sphenoid in the general neighborhood of the sella turcica. As will be pointed out subsequently, this localization should be suspected when signs of meningeal irritation or of frank meningitis develop in cases of sphenoid disease; it is, however, usually unrecognized, and when present is demonstrable only at postmortem examinations.

2. Localized forms of osteomyelitis complicating nasal accessory sinus disease are found as follows:

A. In frontal sinus disease, the following lesions occur:

(*a*) Relatively small areas in the anterior wall of the frontal sinus. These are relatively easily recognized by the swelling over the frontal eminences. The lesion tends somewhat to remain localized and, unless it spreads, gives little cause for concern.

(*b*) Relatively small areas in the bony posterior recess of the frontal sinus. These are more difficult to recognize and most commonly presage a later and more widely spread lesion. Intracranial complications occur, but not as commonly as in the next variety.

(*c*) Relatively small areas in the upper angle of the frontal sinus. These as well as those in group *b* are difficult to recognize clinically, are apt to grow in size and spread rapidly and unlimitedly, are commonly complicated by intracranial lesions (because the diagnosis is made late) and, because of these facts, have a high mortality.

(*d*) A characteristic sequestrum occurs with frontal sinus disease complicated by osteomyelitis of the frontal bone. The sequestrum has the shape of a tennis racket, the narrow part occupying the general region of the internal angular process of the frontal part, the flare spreading obliquely upward and outward in the vertical portion of the frontal bone and lying for the most part to one side of the median line.

In Gerber's series of 29 cases of disease of the frontal sinus the lesion was present in the anterior wall in 13, in the posterior wall in 7, in the floor in 2 and in all walls in 1.

B. With ethmoid disease localized forms of osteomyelitis of the skull bones occur in which the process is more or less limited to the nasal process of the superior maxillary bone, the crista galli and the cribriform plate of the ethmoid or the thin bony plates forming the mesial wall or roof of the orbit.

C. With sphenoid disease the characteristic form of localized osteomyelitis occurs in the posterior part of the body of the sphenoid where articulation takes place with the basilar process of the occipital bone, into which the osteomyelitis also extends.

In the cases differentiated in this group a tendency is exhibited for the lesion to remain relatively localized, and the development of the process is a relatively slow affair which takes months and sometimes years for its final cure or for its complete development. Exacerbations of infection are, however, very common, and in such an exacerbation the tendency toward localization may be lost; then the process spreads with great rapidity over a large part, or all, of the skull, as is characteristic of the unrestricted variety to be immediately described.

3. There is a certain amount of overlapping between the cases in this group and those in the preceding groups, and the physical characteristics of the pathologic process in the cases in this group frequently include those of cases in the preceding groups. Clinically, the manifestations may or may not begin with the milder symptoms of the cases in groups 1 and 2, but the great differentiating factor consists in the fact that the relatively benign characteristics of a localized lesion are lost and the spread of the osteomyelitis becomes unrestricted and extends to the major portion, or to the entire extent, of the cranium. One must distinguish the diffuse form from the circumscribed form of osteomyelitis, which has a tendency to heal. The diffuse form has a tendency to spread over the entire skull and almost always presents an unfavorable prognosis.

Several directions of spread are recognizable, depending on the location of the primary point of infection of the cranial bones. Cases of disease of the frontal sinus show the general tendency to spread upward and outward to the vertical portion of the frontal bone, to the parietal and temporal bones and then backward to the occiput. The ethmoid cases show the general tendency to spread forward and slightly upward to the lacrimal bone, the nasal process of the superior maxillary bone, the cribriform plate and the crista galli. In the sphenoid cases the process spreads backward to involve the basilar portion of the occipital bone. The danger zones are always toward the base of the skull, where an abundance of natural foramina form portals of easy access for the infectious process and where the large sinuses lie in more intimate contact with the affected parts of the bones.

A relative lack of constitutional resistance and a relative extreme virulence of the infecting organism have furnished the explanation why osteomyelitis develops in one patient and not in others under apparently precisely similar conditions. The process may seemingly be unilateral and limit itself to the bones of one half of the cranium or, more commonly, may spread on both sides of the skull. The development of the diffuse form of the osteomyelitis or its extension from more localized forms may occur rapidly, especially in young patients, or, on the contrary, months may be required for the process to evolve.

In Tilley's case, a most extreme one, the process spread upward over the entire vault of the cranium and then downward on each side to, and including, the petrous pyramid.

GROSS AND MICROSCOPIC PATHOLOGY

The gross appearances of the lesions vary with the stage of the disease. In the early stages of osteomyelitis of the cranial bones, the bone presents no abnormalities to the naked eye, although during life the diploe may perhaps have shown some congestion. In the next stage the diploe on section is seen to be markedly hyperemic and to be interspersed here and there with drops of pus. This is followed by the conversion of the medullary tissue of the diploe into granulation tissue bathed in pus, which, on section of the bone, exudes copiously from the cut surfaces. Thrombosed veins may be seen occupying the diploe. In the third stage the purulent secretion, finding its way to the outer and inner surfaces of the bony cranium by the vascular channels or through minute openings formed by the resorption of bone tissue, collects to form abscesses between the bone and the pericranium, on the one hand, and between the bone and the dura, on the other: the para-osseous abscesses.

In the external variety of abscess the pericranium, raised off the surface of the bone by the accumulation of pus, quickly undergoes dissolution, so that the collection of pus comes to lie in the subcutaneous tissues of the scalp. No delimiting abscess wall is formed, but the pericranial abscesses are, nevertheless, when they are multiple, quite discrete and separate from each other. These are the doughy or puffy swellings of the scalp, which form a striking clinical feature of the disease.

The dura shows little or no tendency to break down for considerable periods of time. Larger and smaller collections of pus gather in the extradural space, which, when sufficiently large, may give rise to compression of the intracranial contents. The bone (external) wall of the abscess remains bare, but the dural (internal) surface becomes covered with shaggy granulations. Fine adhesions are usually demonstrable between the deep surface of the dura and the underlying arachnoid.

The fourth stage of the lesion is marked by more or less widespread destruction of the tables of the skull, the external usually giving way before the harder vitreous. By this time more or less extensive areas have undergone necrosis. Sequestration occurs either in large masses, sometimes of characteristic shape, or in small flaky segments. An eroded or wormeaten appearance of the outer table of the skull has given rise to such descriptive terms as "lace-work" (Tilley) or "as if corroded with an acid" (Luc).

During the active stages of the development of osteomyelitis of the skull of this type, new formation of bone, which occurs with such rapidity and exuberance in osteomyelitis of the long bones, either does not appear at all or is scarcely perceptible. On microscopic examination a little attempt at regeneration may be visible, but it is entirely overshadowed by the processes of destruction. Indeed, in this respect, as in many others, the reactive efforts of the tissues seem to be feeble and incompetent. In favorable cases, in which healing and cicatrization become definitely established, however, the gaps made in the skull by the spontaneous or operatively produced loss of tissue are to a large extent or almost entirely filled up again by the formation of new bone.

Microscopically, early specimens show that the first changes consist in an invasion of the bone spaces by micro-organisms, with round cell infiltration of the medullary spaces, the walls of which, at this period, are still quite smooth or are only just beginning to show signs of lacunar absorption. The diploe is hyperemic and congested. Its consistency is decreased, and it contains small fungous growths and droplets of pus. It soon becomes grayish and contains sequestrums. The latter are generally numerous and small, and may remain in the bone for a more or less long time or may be eliminated. The alterations of bony structure are especially seen on the external table.

The later changes, which may be regarded as secondary to the necrosis of the bone, consist in the replacement of the bone marrow by granulation tissue, with absorption, erosion and finally disappearance of the bony trabeculae of the diploe. The outer and inner tables withstand the eroding action until a still later period, so that extensive sheets of granulation tissue develop between the still intact, though lifeless, tables. The blood vessels contain thrombi, more or less infiltrated with pus cells, and signs of perivascular inflammation accompany the thrombosis when it progresses in advance of the general disease of the bone.

The lesions are seldom confined to the bones, but the integuments become inflamed and infiltrated with pus. A case was described by Smith in which a segment of skull with its covering of scalp exfoliated in one piece.

The dura mater is always affected, and becomes thickened and granular throughout the entire extent of the lesion of the bones. If the lesions do not evolve too rapidly, adhesions are formed in the arachnoid, and the brain is protected against encephalitis. But once the natural resistance is overcome, the infection may reach the pia mater and cause any of the varieties of meningitis, or the suppuration may extend to the brain substance and the large cerebral sinuses. The latter seems to be the most common complication.

In cases in which general infection of the blood occurs, the viscera show lesions of pyemia. In long-standing cases the liver may be increased in size and may show amyloid degeneration. The spleen becomes hypertrophied. The lungs show nodules of hepatization and bronchopneumonia.

PATHOGENESIS

The nature of the illness and of its origin in an infection of the nasal accessory sinuses is such as to prevent one from securing specimens of the lesion which would demonstrate the early stages when the transmission of the pathologic process to the bone tissue occurs. Beliefs recorded in the literature regarding the paths and the methods of propagation into the bone tissue are therefore necessarily assumptions based on clinical observations and on pathologico-anatomic evidence obtained from specimens in a late stage of the lesion. In the literature, three methods of infection have been suggested:

1. The infection is described as a direct invasion of the canalicular and medullary spaces of the bone tissue by the organisms of infection from the inflamed area of the lining mucous membrane of the nasal accessory sinus. Preference is given entirely to this method by McKenzie, and only under certain conditions by Schilling (i. e., where the diploic tissue abuts on the wall of the frontal sinus, as, for example, in the neighborhood of the upper recess).

McKenzie is inclined to minimize the part played by the diploe of the bone in relation to the sinus, so emphasized by the continental authors, who contend that the proximity of the diploe to the diseased cavity, or its surgical exposure, lays it open to infection. While he believes that the infection may be able to spread more rapidly once the diploe has been reached, he holds that diffuse osteomyelitis is not limited in its spread to diploic bone only.

2. Another group of observers has described the infection as spreading to the bone tissue by the small veins of the mucous membrane of the accessory sinuses, which in turn communicate with the diploic veins. In Laurens' case, it was possible to demonstrate the thrombosis of the diploic veins far in advance of the maturely developed lesion in the skull. It is generally believed that the unrestricted extension of the osteomyelitis in the cranium is due to this variety of mechanism.

3. McKenzie mentioned a "general advance" of the pathologic process, which, to him, does not seem to select any particular path. As evidence for this conception he cited Wylie's case in which bacteria were demonstrated in the haversian canals under the periosteum, as well as in the diploic spaces and tissues. This suggestion is not difficult to understand. It probably refers to comparatively late cases in which the pathologic process is so widespread in the microscopic picture as

to give the impression that all available paths are being utilized in the spread of the process. This, of course, does not give the true picture.

All three of these methods of spread of the infection are necessarily correct in the observation that the essential part of the process is the spread into the diploic veins. In the light of modern conceptions of disease and of bacteriologic and pathologico-anatomic knowledge, I believe that the following statements may be taken as correct:

There is no clinical or laboratory evidence to show that the occurrence of the osteomyelitis as a complication of disease of the nasal accessory sinuses is ever due to a hematogenous infection, i. e., that a bacteremia first occurs with the primary lesion in the mucous membrane of any of the nasal accessory sinuses, and that the osteomyelitis is a secondary (metastatic) lesion therefrom. The clinical evidence bears abundant proof—and this is borne out by anatomic and histologic studies (Schilling, Laurens, Wylie and others)—that osteomyelitis of the frontal bone or of the skull is due either (*a*) to a spontaneous extension of the pathologic process along contiguous structures and paths, or (*b*) to a direct primary infection of bone tissue during the course of some operative trauma; that is, cases of osteomyelitis of the skull complicating disease of the nasal accessory sinuses are either cases of extension or primary cases of osteomyelitis.

SPONTANEOUS CASES

In the cases in which the osteomyelitis is a spontaneous complication, the mechanism of the extension of the process into the cranial bone must necessarily depend on two factors: (*a*) On the bacteria involved in the infection, with especial reference to the relative preponderance of virulence of the organism and of the opposing strength of natural resistance. This is important because, otherwise, it might be difficult to explain the occurrence of the osteomyelitis in one patient and its nonoccurrence in others, in all of whom all the other factors are seemingly identical. (*b*) On the character of the anatomic structures involved, with special reference to the structure of the osseous tissue and the arrangement of the arterial and venous network.

According to Gray's "Anatomy," the flat bones of the skull are composed of two layers of compact tissue enclosing between them a variable amount of cancellous tissue. These layers are the outer and inner tables of the skull. The outer one is thick and tough; the inner one is thinner, but of denser structure, and more brittle (i. e., the vitreous table). The intervening cancellous tissue is the diploe.

The walls of the frontal sinuses are composed of thin plates of compact bone which make up the anterior, posterior and superior walls of the sinus. Only where the superior and anterior walls meet to form

the superior recess does the compact tissue divide into two tables to include diploe. Just how far the diploe extends into the superior and anterior walls is an individual matter, but only in a very limited number do the anterior and superior walls contain diploe that communicates with the general diploic structure of the flat bones of the skull.

The structure of the irregular bones at the base of the skull is of two varieties. In certain areas, notably where the body of the sphenoid articulates with the occipital, the structure resembles that of other irregular bones, such as the vertebrae: A peripheral layer of compact tissue surrounds a core of cancellous bone. Elsewhere in these bones, the structural masses consist of a series of cellular chambers formed by thin bony plates in which cancellous tissue is for practical purposes absent. At any rate, except at the points indicated there is no direct connection between the cancellous tissue of these bones and the cranial diploe.

These anatomic facts provide the physical basis for the variety of clinical lesions which occur spontaneously as follows:

1. In simple perforation the chances of the infectious process, under pressure of retained pus and insufficient drainage from the sinus, perforating at any other part than through the thin bony platelike walls are relatively few. The chance of perforation in the thin bony plate lying in an area bearing diploic structure depends on the individual distribution of diploe and is relatively slight. This is borne out by clinical observations and anatomic studies: Simple perforation is always seen through the thin bony platelike walls; perforation elsewhere is usually an incident in a much more widely spread area of osteomyelitis; penetration into the diploe is a much more unusual phenomenon and is always the forerunner of extensive areas of osteomyelitis; the relatively greater incidence of this complication in frontal sinus disease than in disease of any of the other accessory sinuses is due to the relative and actual sparsity of physical connection between any diploic tissue in the bony walls of the latter accessory sinuses as opposed to the direct connection of the frontal diploe with that of the rest of the cranium. Osteomyelitis is produced in this way by the mechanical introduction of the infection into the diploe.

2. Other, or more extensive, forms of osteomyelitis occurring spontaneously with accessory sinus disease are due to the extension of a phlebitic lesion along the vascular channels into the diploe.

According to Gray's "Anatomy," the diploe of the cranial bones is channeled in the adult by a number of tortuous canals, which are lined by a more or less complete layer of compact tissue and contain a network of veins. The latter are large and capacious, their walls being thin, and formed only of endothelium resting on a layer of elastic

tissue, and they present at irregular intervals pouchlike dilatations, or culdesacs, which serve as reservoirs for the blood. These are the veins of the diploe; they can be displayed only by removing the outer table of the skull. As long as the cranial bones are distinct and separable, these veins are confined to the particular bones, but when the sutures are united they communicate with each other and increase in size.

As regards the spread of infection by the venous channels, in many instances numerous communicating veins are to be seen with the venous channels in the diploe. Once infection has traveled along these channels into the diploe, the conditions are favorable for an extension of the inflammatory process further along between the tables of the skull. This favorable condition in the diploe for the extension of the infection has been definitely proved pathologico-anatomically by several observers (Schilling, Laurens and others). The older the patient, the less barrier there is to the extension of the infection, and, as McKenzie and others have pointed out, in adult life there is a direct continuity of osseous structure between the contiguous bones of the skull and the diploic veins that traverse the sutures of the skull. Diffuse and rapidly spreading forms of osteomyelitis must necessarily always occur by way of the vascular channels, as once the infection has penetrated into the vascular network no efficient obstacle is present to the further spread of the infection along the veins past the sutures and from one cranial segment to the others.

POSTOPERATIVE CASES

It would be well to understand at the outset, that although the literature speaks at length of cases in which this complication occurs spontaneously, this probably occurs in a much smaller percentage than is apparent, for the term "operation" should include not only those large manipulations ordinarily dignified by the attachment of some surgeon's name, but also all of those minor manipulations (extractions of polypi, etc.) which are not classified as such, but are called "treatment" of one kind or another and are commonly performed in the practitioner's or specialist's office. These, under proper circumstances which are all too commonly present, can be productive of all grades of traumatism to the soft parts and to the vascular channels.

In spite of the fact that in the literature the total number of cases in which osteomyelitis of the skull occurs as a complication of nasal accessory sinus disease is approximately 3 per cent, the opinion has become prevalent among otolaryngologists that while osteomyelitis of the skull bones is one of the uncommon spontaneous complications of nasal accessory sinus disease, it is one of the commonest serious sequelae of operation for the cure of the latter. This statement seems to hold true for the most part in cases of frontal sinus disease. It holds true

to a much less, and possibly to a negligible, extent in cases of ethmoid disease, and not at all in cases of sphenoid or antral disease. The facts in the matter seem to be as follows:

Gerber's communication listed 29 cases of osteomyelitis of the frontal bone or skull complicating accessory sinus disease. In 13 of these cases no definite etiology was mentioned; in the other 16, influenza, syphilis, trauma, coryza and measles were cited as etiologic factors. I personally believe that such etiology did not play a rôle in the production of the osteomyelitis, but rather in the production of the original accessory sinus disease. The important point is that among the 16 cases cited by Gerber, osteomyelitis of the skull followed some operation on the accessory sinuses 10 times, a very large proportion.

McKenzie's statistics cover 41 cases of osteomyelitis of the frontal bone; the complication developed spontaneously in 21 cases and after some form of operation in 20.

Ropke considered as instances of postoperative infection no less than 9 of the 16 cases known to him.

In the Mount Sinai group practically all of the cases of osteomyelitis of the skull complicating disease of the nasal accessory sinuses developed prior to the performance of any operation of the Ogston-Luc or Killian types in the hospital. There is a possibility that in some of them, at least, some minor forms of "office" operation had been done previous to the admission of the patient to the hospital. In general the osteomyelitic process was discovered at the time the first operation was done in the hospital. This experience corresponds with that of Burger.

This discrepancy seems to offer insuperable obstacles to adjudication except on the basis of individual interpretations of operative findings. My personal impressions incline me to agree with those who believe that operation is a frequent precursor of the osteomyelitic complication. An examination of all the factors entering into the various types of operation for nasal accessory sinus disease seems, therefore, in place.

A. There are two types of operation: (*a*) Operations of the Ogston-Luc type consist in opening the frontal sinus through its anterior wall, in carefully curetting its anterior portion and in establishing a large communication between the sinus and the nose, at the same time destroying the anterior ethmoid cells in the region of the nasofrontal duct. Drainage into the nose is insured through the nasofrontal aperture, and the operation is completed by immediate suture of the cutaneous incision.

Probably no other type of operation has been more generally performed on the frontal sinus, yet because of anatomic structure, clinical evidence has been much against the performance of an operation that

does not permit of thorough inspection of the entire sinus and of the ethmoid cells. In order to bring this about, it is necessary to remove the whole of the anterior wall of the sinus, and also to a greater or lesser extent the floor. By such a procedure, one is able to inspect the whole interior of the cavity thoroughly, however large it may be; no recess can escape one's observation; the septum between the two cavities can be examined, and the presence of extension of the ethmoid cell into the orbital roof will thus not be overlooked.

(*b*) This is the reason and the basis for the modern Killian type of operation. The whole sinus, with its recesses and partitions, is thoroughly inspected, and the cavity is almost completely obliterated by the resection of its interior and inferior bony walls. Further, by the removal of the ascending or frontal process of the superior maxilla, excellent access is obtained to the ethmoid cells, and a large opening of communication is thus made between the frontal sinus and the nasal cavity, so that good drainage into the nose can be established.

B. Two factors are constantly present in all cases in which operation is done for nasal accessory sinus disease:

(*a*) Infection is always present, and the operation must necessarily be done through infected territory. The only possibility of preventing this would be to arrange the steps of the operation so as to remove the entire infected area in one segment, a procedure that, while possibly technically feasible, would be unwarranted for any and all other reasons, would result in a deformity of contour that would not be acceptable to either the surgeon or the patient and would probably yield an enormous mortality. One must content oneself, therefore, with less radical measures—a state of affairs that makes it technically impossible to remove every bit of infected tissue. Foci of infection are, therefore, necessarily left which readily contaminate the newly exposed bone tissue in the operative field.

In those unfortunate patients in whom operation is undertaken for the cure of disease of the sinuses, the inflammatory condition is markedly enhanced during the operation. The granulation tissue in the sinus, which has hitherto formed a barrier to the invasion of the organisms, is broken down and a path is opened, which readily admits of their further spread into the frontal bone.

(*b*) The second constant factor is operative trauma. The same anatomic studies to which I alluded previously show also why operation for sinusitis is so apt to be complicated by osteomyelitis of the skull. If the operator is not careful, or if the anatomic distribution of the diploe is extraordinarily well developed and reaches beyond the usual limits, opening into the diploe by chisel or curet is to be expected and does commonly occur. Infection is then implanted directly in the diploic contents.

An analysis of the cases by Turner showed that in 74 per cent of those in which the osteomyelitis appeared, the complication occurred after opening and draining the sinus (the Ogston-Luc type of operation). In 88 per cent of these, death followed a single operation, while in the remainder more than one operation had been performed, owing to the failure of the first to effect a cure of the suppurative condition. The remaining 24 per cent of the cases all proved fatal after an operation for obliteration of the sinus; in 50 per cent of these, simple opening and draining had been previously performed without success, the more radical method having been a secondary procedure. It is evident, therefore, that a considerably larger proportion of the cases occurred after the Ogston-Luc type of operation than after the operation of obliteration. This general impression was confirmed by Burger.

I am unable to make any comparison as regards this phase of the discussion with the experience at Mount Sinai Hospital, as there practically all the cases of osteomyelitis of the skull complicating nasal accessory sinus disease occurred spontaneously.

Consideration of the steps in either form of operation gives an adequate explanation for this state of affairs. In the simple (Ogston-Luc) operation an opening is made into the frontal sinus through its anterior wall. The density of the bone forming the anterior wall of the frontal sinus varies considerably, and my anatomic studies have shown that this general area is the one in which the diploic structure is commonly found and in which, when the diploic structure is exaggerated, it spreads downward for a considerably increased distance. The more the diploe of the walls of the frontal sinus is developed, the greater is the anatomic predisposition for the development of osteomyelitis of the frontal sinus. For this reason, youth is more predisposed than mature age. In the former, the diploe has larger meshes and has a larger supply of vessels than in the latter. Cases in which prior to and during the first operation on the frontal sinus nothing pointed to a disease of the bone, and in which such a disease occurred only after the operation intensify the feeling that the operation itself is the cause of the infection of the diploe.

The mere chiseling of the anterior wall of the frontal sinus is sufficient to open and to infect the diploe of the frontal bone. This was probably so in the case of St. Clair Thomson and in that of Luc and Lermoyez.

In the radical operation (Killian type), the complication is not so likely to occur, because one of the points of this type of operation is the retention of as much of the anterior wall of the sinus as is possible, the approach being made through the floor in order to prevent the deformity that accompanies excessive removal of the anterior wall.

The floor of the sinus practically never contains diploe, the compact bone tissue of the bony plate does not take up the infection so readily, and the complication therefore appears less often after this type of operation. As a matter of fact, in many of the cases in which this radical type of operation has been performed and in which the osteomyelitis developed it has been preceded by the simple drainage (Ogston-Luc) type of operation.

Once infection is introduced into the diploe, the physical basis for the localization, the firm lodgment and the spreading of the infectious process, depends on the character of the vascular network of the skull and on whether the infection is lodged in (1) the venous or (2) the arterial plexus.

1. In the former a thrombophlebitis becomes established in the venous plexus of the diploe. The character of the osteomyelitis, whether localized or diffuse, the relative rapidity of spread and any other physical characteristics depend on the area of venous plexus involved in the thrombophlebitis and on the biologic virulence of the infecting organism as opposed to the natural protective powers of the host.

2. In the second group an injury most probably takes place, during the course of the operation, to an arterial trunk and a secondary thrombosis occurs in the latter.

According to Gray's "Anatomy," the supra-orbital artery arises from the ophthalmic as that vessel is crossing over the optic nerve. Ascending so as to arise above all the muscles of the orbit, it passes forward, with the supra-orbital nerve, between the periosteum and levator palpebrae, and, passing through the supra-orbital foramen, divides into a superficial and a deep branch, which supply the integument, the muscles and the pericranium of the forehead, anastomosing with the frontal, the anterior branch of the temporal and the artery of the opposite side. This artery in the orbit supplies the superior rectus and the levator palpebrae and sends a branch inward, across the pulley of the superior oblique muscle, to supply the parts at the inner canthus. At the supra-orbital foramen it frequently transmits a branch to the diploe.

The ethmoidal branches consist of the posterior and the anterior. The former, which is the smaller, passes through the posterior ethmoidal foramen, supplies the posterior ethmoid cells and, entering the cranium, gives off a meningeal branch, which supplies the adjacent dura mater, and nasal branches which descend into the nose through apertures in the cribriform plate, anastomosing with branches of the sphenopalatine. The anterior ethmoidal artery accompanies the nasal nerve through the anterior ethmoidal foramen and supplies the anterior ethmoid cells and frontal sinuses; entering the cranium, it gives off a meningeal branch, which supplies the adjacent dura mater and nasal branches, which

descend into the nose through the slit by the side of the crista galli and, running along the groove on the under surface of the nasal bone, supply the skin of the nose.

The frontal artery, one of the terminal branches of the ophthalmic, passes from the orbit at its inner angle and, ascending the forehead, supplies the integument, muscles and pericranium, anastomosing with the supra-orbital artery and with the artery of the opposite side.

The nasal artery, the other terminal branch of the ophthalmic, emerges from the orbit above the tendo oculi and, after giving a branch to the upper part of the lacrimal sac, divides into two branches, one of which (the transverse nasal) crosses the root of the nose and anastomoses with the angular artery; the other, the dorsalis nasi, runs along the dorsum of the nose, supplies its outer surface and anastomoses with the artery of the opposite side and with the lateral nasal branch of the facial.

Injuries to any of these major arterial channels are accountable for those larger, more or less anatomically well defined areas of osteomyelitis which result in correspondingly similar masses of sequestrated bone. One of these typical clinical entities was alluded to previously in this communication as occurring in the frontal bone. The physical basis for this is undoubtedly injury to the terminal branch of the frontal artery after its emergence through the supra-orbital foramen, which branch supplies, among other structures, the pericranium of the vertical portion of the frontal bone just above the glabella. An arterial lesion of this kind undoubtedly also supplies the physical basis for the lesion described by Smith in which bone and integument exfoliated in one piece. Another example of this type of lesion is the sequestration that occurs involving the crista galli and cribriform plate of the ethmoid, for which the physical basis is injury to the ethmoidal arteries.

Operative injury by itself, whether to the venous plexus of the diploe or to a major arterial trunk, would possibly not be sufficient to give rise to osteomyelitis of the skull. The constant presence of virulent organisms, both normally residing in the nasal and nasal accessory cavities and also present as the inciting agent of the original sinusitis, provides the necessary and essential element for the establishment of an inflammatory lesion in the bony tissue. The operative injury provides a soil of decreased resistance for the growth and development of the organisms and in the cases of injury to an arterial trunk establishes areas of bone in and from which nourishment by blood supply has been eliminated. Operative injury plus infection provides the factors necessary to give the total clinical picture.

C. The question of drainage after operation is also an important item. In neither of Lack's fatal cases was the mucous membrane of

the sinus curetted; yet an osteomyelitis of the frontal bone supervened, and death followed from intracranial complications. Tilley blames defective drainage into the nose, coupled with complete closure of the cutaneous incision, as the cause of disaster in these cases. Most surgeons will probably agree with the necessity of establishing free drainage, a point that has from time to time been insisted on by the majority of writers on the subject. In 2 fatal cases mentioned by Turner no sutures were placed in the incision, thus insuring free superficial drainage; in one of these, however, the fatal result was attributed to defective nasal drainage; in the other, numerous ethmoid cells extending into the orbital roof were overlooked, and bone sepsis was thus set up.

I think that Turner's experience is almost sufficient to show that the question of the drainage itself is not of much importance as regards the appearance of the osteomyelitis. Under such circumstances the latter probably should be explained on the bases discussed in the section devoted to the spontaneous occurrence of this complication after nasal accessory sinus disease.

SYMPTOMATOLOGY AND CLINICAL COURSE

1. *Cases of Simple Perforation.*—In cases of simple perforation, through the floor of the sinus, the symptomatology is simple and a diagnosis is easily made. In a patient with symptoms of frontal sinus disease, the orbital fissure narrows, the orbital contents become swollen, and there is a good deal of tearing. Pain is present or becomes exaggerated. There is usually no fever. Frequently a small mass, a subperiosteal abscess, is found on the roof of the orbit. If it is not promptly incised, and if it should rupture into the orbit, a diffuse orbital phlegmon develops.

In cases of simple perforation through the anterior wall, the symptomatology is also not difficult, the region of the glabella becomes swollen and edematous, and an abscess forms under the skin which either ruptures spontaneously or is incised. In either case, a probe quickly demonstrates an opening leading into the frontal sinus.

The symptomatology of cases of ethmoid perforation is nil when the perforation occurs in the nasal cavities except for some usually undervalued increase in the nasal discharge. In cases in which the perforation occurs into the orbit, the symptomatology is that characteristic of an orbital phlegmon, is sometimes more or less dramatic and is frequently secondarily complicated by intracranial lesions.

The symptomatology of cases of sphenoid perforation is as follows:

(*a*) Perforation in the floor of the sphenoid is sometimes merely marked by an undervalued increase in the nasal discharge. If the

mucous membrane remains intact, a swelling can be palpated in the roof of the nasopharynx; the diagnosis should be made when such a swelling is present. Occasionally the perforation is to one or the other side of the midline; suppuration then develops in the general neighborhood of the pterygomaxillary fossa. The latter condition may be suspected if, with distinguishable signs of nasal disease, difficulty and inhibition are present in the free use of the temporomaxillary articulation (opening of the jaws). Commonly this complication is unrecognized.

(*b*) Perforation in the roof of the sphenoid should be suspected when, in a patient with sphenoidal disease, signs of an intracranial complication (especially meningitis) appear. Headache in the top of the head is a common prodromal symptom.

2. *Cases of Osteomyelitis of the Skull of Ordinary Type: Localized Form.*—In the ordinary case of an insidious development of osteomyelitis complicating a frontal sinusitis the suspicion that the osteomyelitis is developing does not arise for an appreciable interval. In patients who have not been operated on, clinically, the most important symptom is the extension beyond the borders of the frontal sinus of an edematous swelling combined with an actual doughy infiltration that extends below the scalp and remains constant when there is free drainage of the pus. Multiple subperiosteal abscesses speak for osteomyelitis especially if there are concurrent general symptoms. This applies the more so when the probe below the galea encounters everywhere rough bone. Morel and Hubert compare the osteomyelitis bone with "rotting wood."

If an operation on the frontal sinus has already preceded, the beginning of the osteomyelitic complication shows itself first in a reddening, swelling and loosening of the scar. The lifting up of either wound flap reveals pus. The probe reveals the bone conditions just described. These symptoms indicate the necessity for a new operation.

At the secondary operations the wound is enlarged by means of new incisions. The skin is found to be swollen and infiltrated and to contain islands of suppuration. The friable and necrotic bone is removed. The subadjacent dura mater is fungous and purulent. The limits of the osteitis are not grossly distinguishable and are reached with difficulty. The bone is always thickened, soft and red. If all of the affected portions are removed, the temperature descends and all the symptoms disappear. But more frequently there is only a period of calm, and on the second or third day after the revision the wound suppurates. Cicatrization does not take place, the gangrene extends, and the temperature becomes irregular. A new operation is again indicated, and is then usually performed in an attempt to get beyond the diseased area.

The same clinical picture may be reproduced three or four times at more or less long intervals, and a cure may finally be obtained. But in most cases the pain is continuous, and the swelling of the wound persists.

In the severe, rapidly progressing cases, this always occurs with increasing frequency. New subperiosteal abscesses form. Frequently in the severe cases the local symptoms decrease, but the general symptoms increase and the entire organism is invaded by a general infection of the blood. There is severe intoxication of the organism, with chills, profuse perspiration, delirium and remittent fever. There is an increase in the size of the liver and spleen. Death occurs from extension of the infection to the brain, sinuses or lungs, regardless of new operations.

The cases in groups 3 and 4 occur for the most part in cases originating in frontal and ethmoid disease.

3. *Diffuse Form of Osteomyelitis of the Skull.*—In the diffuse form there are certain symptoms that are more or less characteristic. The pain is diffuse, and the patient cannot localize it. The headache generally is more severe at night, as is the case with all pains of osseous origin. The patient is unable to sleep, and even large doses of opium or of other narcotics do not relieve the pain. The pain is generally more marked at the beginning of the disease, but is variable. In some cases it does not appear until the end, and it may even be absent.

The swelling around and about any previously made wound is not sharply defined. It may be of a doughy nature, and then it may make one think of an erysipelatous infection. As the process spreads, discrete swellings appear at some little distance; these are subperiosteal abscesses. The skin is red and very tender.

Fever is for the most part continuous, although the curve may fall to normal limits for short intervals, only to return promptly. At the onset the temperature is apt to be high and have an irregular course. During the course of the disease, when the manifestations are particularly severe, the temperature frequently assumes the characteristics customarily seen in typhoid fever. Chills are not common; they occur only in cases of general infection, frequently indicate the presence of a complicating phlebitis of one of the intracranial sinuses, are present with and without positive blood cultures and are generally of unfavorable prognostic value. There may be terminal hyperpyrexia.

4. *Severe Forms of Osteomyelitis of the Skull.*—Hyperacute cases of osteomyelitis of the skull complicating nasal accessory sinus disease are relatively rare. The clinical picture of the cases in this group is that of a profound general infection: Marked toxemia is present, and there is a severe organic reaction. The disease begins with violent chills, and high fever follows; the general condition frequently and

suddenly becomes very poor; the pulse becomes weak and feeble; the urine is scanty and sometimes contains albumin; diarrhea often appears; the patient is prostrated, and is covered with perspiration. In the most severe cases death follows in a few hours from the general infection.

In the very sick patients, alarming secondary intracranial complications develop and overshadow the symptomatology of the osteomyelitis. These will be discussed subsequently and more extensively on another occasion.

A general blood infection (sepsis, septicemia, pyemia) does not commonly take place, as demonstrated by positive blood cultures. Nevertheless, transient or more prolonged states of blood infection undoubtedly occur, as in some of the fatal cases lesions are described and are present in organs distant from the seat of the local lesion or its neighborhood, the mechanism for the production of which is surely blood-borne.

Positive blood cultures are not the rule in this form of osteomyelitis of the skull. This is another proof that this form of osteomyelitis is not a hematogenous infection. In the presence of some other complications, notably meningitis, positive blood cultures may be expected.

CLINICAL COURSE

The course of the disease is generally protracted, and is frequently interrupted by intervals of relative euphoria. In Luc's case there was a period of three months between the last manifestation of osteomyelitis and the first one of pachymeningitis; the entire disease lasted two years; a cure was then obtained which Luc ascribed to the slight virulence of the streptococci. In general, the development of the entire disease and its clinical picture may take as little as two or three months, or as long as two or more years. In the fatal cases the cause of death is much more commonly the effects of the intracranial complications; much less commonly it is the effect of a general blood infection.

COMPLICATIONS

In cases of general blood infection (sepsis, septicemia, etc.) metastatic lesions are found post mortem in the various organs of the body; they are especially common in the kidneys, the liver and the lungs. I have encountered no mention of an endocardial lesion in cases of this kind (i. e., cranial osteomyelitis) in the literature. As was referred to previously, the relatively uncommon occurrence of a general infection makes the finding of such metastatic lesions relatively infrequent in clinical practice. Metastatic lesions such as these (i. e., of the kidney, liver or lungs) give no dominating picture and are lost in the general symptomatology, both subjective and objective, of the clinical picture.

Localized abscess formation is a relatively common complication. The types commonly found include: A. In disease of the frontal sinus, (*a*) various forms of abscess of the scalp (subperiosteal abscess of the bones of the skull) and (*b*) orbital abscess and phlegmon occur.

B. In the ethmoid cases the common complication of this type is orbital abscess. Orbital complications of nasal accessory sinus disease are more common than endocranial complications, which they frequently precede. There seems little doubt that the majority of intra-orbital inflammatory conditions owe their origin to disease of the nasal accessory sinuses. This is especially so for the cases of frontal and ethmoid disease. Birch-Hirschfeld's experience is that 60 per cent of orbital inflammatory conditions are due to disease of the nasal accessory sinuses. Kuhnt thinks this percentage too low.

The mechanism is as follows: 1. By perforation through the bony wall. This is especially common with frontal and ethmoid disease. 2. By direct extension of the process through some natural opening—optic foramen, superior orbital fissure, etc. The autopsy on a patient of Leegaard's showed pus in the sphenoid sinus, in the orbit, around the first branch of the trigeminal and around the chiasm. 3. By the thrombosis of the ophthalmic veins. An illustrative case is the following: The autopsy in Vail's case showed chronic sphenoid suppuration (right), necrosis of the orbital wall of the sphenoid, orbital phlegmon and suppurative phlebitis of the ophthalmic vein, the cavernous sinus, etc.

C. In the sphenoid cases, (*a*) abscess in the roof of the nasopharynx, a variety of, and a frequent predecessor of, frank retropharyngeal abscess, can be palpated in the posterior wall of the nasopharynx; (*b*) abscess in the pterygomaxillary fossa is observed. These abscesses most commonly develop as a result of suppurative conditions in the base of the skull associated with sphenoid disease and are usually associated with thrombophlebitis of the veins coming from the cavernous sinus and passing into the superior nasopharynx. Pterygomaxillary abscess is especially common in those cases of sphenoid disease in which a tonsillitis or a tonsillar operation has precipitated the clinical picture. The symptomatology has already been discussed.

Except for these conditions, osteomyelitis of the skull is especially noted for the occurrence of intracranial complications. In the 29 cases of osteomyelitis of the skull collected by Gerber there developed:

Pachymeningitis	4 times
Extradural abscess	11 times
Intradural abscess	1 time
Thrombophlebitis	5 times
Cerebral abscess	4 times
Meningitis	9 times
Meningo-encephalitis (1 septic case)	3 times

In 1895, Kuhnt, in his work on the frontal sinus, collected 17 cases of intracranial complication associated with suppuration in that cavity. In 12 of these the frontal sinus only was diseased, while in the remaining 5 there was associated suppuration in other accessory cavities. Dreyfuss, in his monograph published in 1896, reported 19 cases of a like nature following suppuration of the frontal sinus. The latter, however, are not to be regarded as additional to those reported by Kuhnt. Killian, in his article on the frontal sinus in Heymann's "Handbuch der Laryngologie," published in 1900, brings the total number of frontal sinus complications up to 30. Since that date Turner has been able to add 12 cases not included in the statistics of the aforementioned writers, and Eagleton, Kramer, Burger and others have since added many more.

According to Turner, the postmortem evidence of intracranial infection is somewhat meager. Newton Pitt, who gave an analysis of 9,000 autopsies, found only 1 case of cerebral abscess due to nasal suppuration; the patient had had nasal polypi and ethmoid disease. Gowers considered that chronic nasal disease is an occasional but rare cause of cerebral abscess. He stated that in 240 instances of abscess of the brain, the nose was the primary seat of infection in 6, but the sinuses implicated were not specified. Treitel found in 6,000 postmortem examinations 3 cases in which the intracranial disease had its origin in the nose, namely, 2 of cerebral abscess and 1 of purulent meningitis. Wertheim had access to the records of 10,394 autopsies; among these were 127 cases of endocranial suppuration. Fourteen were stated to be of nasal origin, but in 6 only was the exact mode of infection specified. In only 3 of these, again, was the frontal sinus the probable primary source of the complication.

So far as Turner has been able to ascertain, these are the main facts at one's disposal, and therefore, unfortunately, I cannot draw any definite conclusions as to the percentage of cases of suppuration of the frontal sinus in which intracranial complications arise. That they do occur, there is ample evidence to show; indeed, it seems that the reason for most of the case reports in the literature is the occurrence of these complications, and there is the possibility that they may even occur more frequently than the experience described would indicate.

I am inclined to agree with Burger that the majority of endocranial complications after nasal accessory sinus disease occur through the mediation of an osteitis of the bones housing the sinuses and of their continuations into the adjacent flat cranial bones. According to Burger this is based on experience at operation and autopsy. Dreyfuss found this status in 12 of 19 cases in relation to the frontal sinus. Gerber was outspoken in his belief that almost every endocranial complication has its origin in infection of the bony wall.

The transmission of the infection occurs through the anastomoses between the veins of the diploe and the veins of the dura. At the parietal bone it eventually occurs through the parietal emissary vein. Burger's interpretation is that the infection of the mucous membrane is communicated to the underlying bone and then transmitted further.

The development of the intracranial complications and the clinical course during their development have in most instances followed a fairly definite order. The first stage is comprised in the infection of the skull bones and in the development of the localized or, more commonly, diffuse form of osteomyelitis. In some of the cases the bone lesion is localized at the time the intracranial complication occurs; in others the osteitis has become diffuse, and in rare instances few or insignificant changes can be discerned.

The second stage is marked by the occurrence of an external pachymeningitis; an extradural abscess usually results.

The third stage is a progression of the second and, according to most of the observers, is produced fairly rapidly. A subdural infection occurs, and arachnoiditis and meningitis result.

In the further spread of the infection beyond these limits the process spreads to the intracranial venous sinuses and to the brain. Thrombosis of the large sinuses, especially the cavernous sinus and the superior longitudinal sinus, is commonly seen in the published reports of cases. Abscess of the brain may occur at any stage of the development or in the absence of many of the stages herein outlined.

The majority of the intracranial complications occur in accordance with the following mechanisms: (*a*) by contact because of contiguity of structure, (*b*) by vascular and lymphatic connections and (*c*) by contact with a secondary complication, usually a discrete extradural abscess separated and at a distance from the region of the main focus of cranial osteomyelitis.

PROGNOSIS

The prognosis varies according to the form of the disease. The prognosis is generally benign when the osteomyelitis is confined to one bone.

All cases of diffuse cranial osteomyelitis after disease of the nasal accessory sinuses have terminated in death unless surgical treatment was instituted. An immediate and extensive operation may limit the process, but some cases terminate in death even after many operations have been performed. There are always cases in which it is impossible to reach the limits of the infection, and the chances of cure are slight in these instances. Extension toward the base of the cranium or toward the petrous bone increases the gravity of the prognosis. A streptococcic infection is very serious and is frequently accompanied by endocranial complications.

TREATMENT

The essential treatment can be divided into three stages:

1. The stage of prophylaxis. Although osteomyelitis of the skull may occur spontaneously with nasal accessory sinus disease, the available information shows conclusively that operation on the sinuses is the most important single item in the production of this much to be feared complication. Hence, it behooves every one never to operate in cases of accessory sinus disease unless the indication is a most dominating one. I am sure that a little care will obviate many operations that are now customarily done.

2. The stage of uncomplicated osteomyelitis of either the frontal bone or the skull. Once osteomyelitis has set in, the only chance of saving the patient lies in the immediate and entire removal of the diseased bone. The superficial area of the bone resected must exceed the obvious limits of the disease. The resection must be radical, and in doubtful areas it is safer to remove bone than to leave it. Obviously under these circumstances success will follow one's efforts more often in the localized than in the diffuse form of the disease owing to the fact that radical operation will be more often technically feasible in the localized form. The usual experience is that the need for revision is frequent and imperative.

3. Subcutaneous abscesses under the scalp. These should be incised and drained as soon as they are discovered. Even when multiple and extensive, they do not necessarily determine the end-result, as this depends on all the other factors associated with the osteomyelitis.

4. The stage with intracranial complications.

(*a*) Extradural abscess is usually a relatively simple matter when no other complication exists; incision and drainage are indicated. It is important to make the diagnosis early so that drainage can be effected before the infection spreads inward to the meninges. In the presence of other complications, the prognosis depends on the latter.

(*b*) Serous meningitis usually takes care of itself and needs no active therapeutic measures. Its presence, however, should make one look carefully for other intracranial lesions. Under appropriate conditions, the demonstration of a serous meningitis might indicate that there is a local focus of osteomyelitis in the bone which needs operation or revision, and when all other conditions are equal and favorable, the indication should be met promptly.

(*c*) Purulent meningitis is almost always a fatal lesion when it complicates an osteomyelitis of the skull. Little if anything can be done in a therapeutic way, beyond releasing intracranial pressure either by repeated lumbar puncture or by drainage of the cisterna, and beyond attempting to combat the infection by the introduction of antibacterial

serums of one kind or another. All supportive measures and the usual forms of treatment are, of course, in order. The prognosis is extremely poor, and there is the usual huge mortality.

(*d*) The usual experience with abscess of the brain applies here also. Localization having been made as soon and in as early a stage as possible, craniotomy should be done and the abscess efficiently drained for a sufficiently long time. In the neurologic surgical literature there seems to be a little improvement in the number of successes after operation for abscess of the brain. But, unfortunately, under the conditions of a chronic osteomyelitis of the skull, and with other intracranial complications coexisting with the abscess of the brain, the outlook is unfavorable and a huge mortality is to be expected.

(*e*) Thrombosis and thrombophlebitis of the intracranial venous sinuses are, for practical purposes, impossible lesions for therapy in the present state of knowledge, even though I have come across several isolated cases of recovery after operation.

MORTALITY

A considerable number of deaths have followed operation on the frontal sinuses because of the occurrence of osteomyelitis of the skull and of secondary complications within the cranium. The fatal issues have resulted from the intensity of the local lesion, its persistence and noneradicability and the tremendous facility and ready opportunity for the establishing of other secondary complications, any one of which increases enormously the chances for a fatal issue of the total clinical entity.

No attempt will be made in this communication to establish the percentages of cases "cured" or "not cured" or of the fatalities, as it is impossible to do this accurately for several reasons: (1) The number of cases of disease of the frontal accessory sinus that actually occur is undoubtedly much greater than the number reported; (2) human nature runs true to form in this as well as in other things, and one likes only to report successes, and (3) patients often are discharged temporarily from one hospital to obtain advice at other institutions, thus tending to complicate the statistics. Some idea may be had, however, of the true mortality from the statistics compiled by Gerber: Of 29 cases, none was cured and 20 terminated fatally.

SUMMARY

The clinical picture presented by the majority of these cases is very similar. The infection that leads to the fatal result is usually postoperative, and occurs within two or three days after an operation for disease of the nasal accessory sinuses. The diploe of the frontal bone is invaded by the organisms, and an osteomyelitis is set up. The

infection spreads thence, on the one hand, through the outer table, giving rise to subperiosteal abscesses beneath the scalp; on the other hand, it passes inward, and may subsequently give rise to extradural or subdural abscess, general meningitis, cerebral abscess or a thrombosis of the longitudinal or other large sinuses.

The dura mater is affected in practically all cases, and the pachymeningitis may remain localized a long time. Thrombosis occurs chiefly in the lateral or longitudinal sinuses. The invasion of a large venous sinus is often manifested by emboli with distant metastases. Pneumonia or bronchopneumonia frequently occur.

It is generally easy to make a diagnosis of osteomyelitis in the bones near the orbit or ear. However, it is often difficult to recognize the diffuse form because the general symptoms are often more pronounced and may mask the local symptoms. A diagnosis must be made before operation if a careful study is made of the symptoms that accompany the sinusitis or mastoiditis.

When the osteomyelitis becomes evident, it is necessary to operate, and the operation should be as extensive as possible. The cranial bones should be resected beyond the limits of the lesion. If the wound continues to granulate and if the temperature remains high, sequestrums are present and they must be removed as completely as possible.

There is a large mortality. The patient succumbs to one or more of these conditions with or without the manifestations of a general blood infection, as evidenced by the presence or absence of a positive blood culture, after a varying number of weeks or months ranging to one or more years.

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A GRAPHIC METHOD AND CHART FOR HEARING TEST WITH TUNING FORKS

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BROOKLYN

The practical advantages of a graphic record of air-conducted sound tests of hearing acuity will at once be obvious to the otologist. Herewith are presented a method and chart for this purpose. The actual testing and charting are so simple that it requires but a few moments to train a nurse or clerk for the procedure. A little experience with the method will permit the observer to glean from a glance at the graph a close approximate of the hearing acuity recorded.

The advantages over the audiometer test are: absence of an admixture of bone conduction, portability and inexpensiveness. The audiometer, of course, has the scientific superiority of a wider and continuous range.

GENERAL PRINCIPLES

1. The greatest sound intensity of a vibrating tuning fork occurs synchronously with the striking of the fork.

2. The more forcible the striking of the fork, the longer will be the period of vibrations. The period, from the striking of the fork until the point of lost audibility in the normal examining ear, will be called the period of diminishing vibrations.

3. From the foregoing statements it is axiomatic that there is a constant ratio between the force of the blow, the intensity of the sound and the length of the period of diminishing vibrations. Therefore intensity can be measured in terms of the number of seconds (in the period of diminishing vibrations) from any particular point of intensity or loudness that one may wish to record. That point of tuning fork intensity is to be recorded which is the last audible to the impaired ear under examination. The interval from this last point to the point of lost audibility in the normal ear of the examiner will be termed the acoustic differential.

4. By determining and charting the acoustic differential for each of a series of forks, a record is obtained of the hearing acuity relative to the normal ear, which is all that is required for clinical purposes.

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From the Neuro-Otological Clinic, Brownsville and East New York Hospital.

In the charts it will be noted that a separate graph is used for each ear; that each pitch column includes two lines, the right one for the markings of the original test and the left for a test directly after inflation, if this is desired, and that the numerals to the left represent seconds. The double line across the top of the pitch columns is the denominator of normal hearing. The double angular line *B. T. D.* (borderline of total deafness) will be explained presently. The double line in the lower margin receives the markings for total deafness under the respective pitch column.

Suppose the patient is now to be tested. I strike fork *c* 32 with moderate force and hold it one-half inch (1.27 cm.) from his left ear;

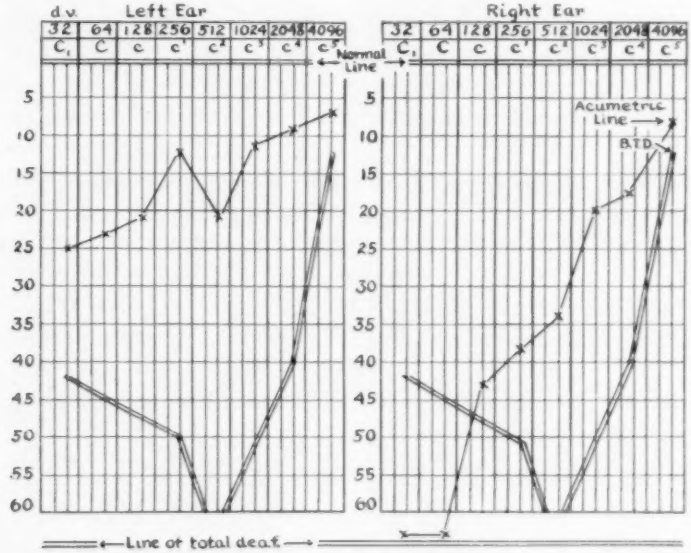

Chart 1.—Graphic record in a case of arrested chronic suppuration with dry perforation in both ears. The left ear shows a moderate impairment with slightly more than half the acuity range lost for C_1 and also for c^5. The right ear shows total deafness for C_1 and C and marked impairment for the whole series of forks.

in my other hand is a stop watch which I shall start when the patient announces the instant of lost audibility. The patient announces this point, the watch is started, and the still vibrating fork is held one-half inch from my (normal) ear: I wait for the point of lost audibility, then stop the watch and read off (in this case) twenty-five seconds, which is recorded in the proper place on the chart. This process is repeated for the remaining forks, and the marks are connected with straight lines. The result is the acumetric line.

As this record now stands, it might give a false impression to the examiner who is unaccustomed to this method. For instance, at c^5 the

acoustic differential of seven seconds might easily be visualized as a slight impairment. But, if he knows that c^5 fork, by the very nature of its construction, cannot be made to vibrate longer than twelve or thirteen seconds from the point of highest intensity, he will see that an acoustic differential of seven seconds is a marked defect. On the other hand, a c^2 fork has the characteristic of possessing the longest vibration period of all the forks, up to eighty seconds, or about six times as long as that of c^5. It would therefore help the eye if these vibration time limits were fixed on the chart by a line crossing the proper levels of each pitch column. The examiner accustomed to reading these

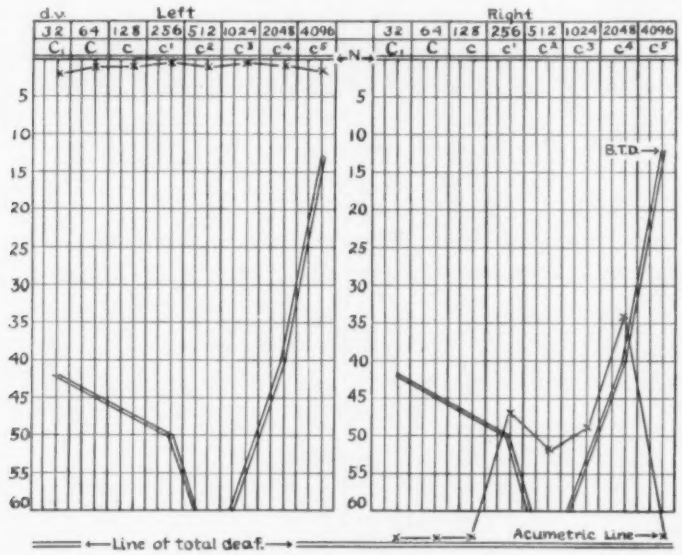

Chart 2.—The left ear shows normal hearing; the right ear, total deafness for forks C_1, C, c and c^5 and almost total deafness for c^1, c^2, c^3 and c^4.

graphs does not require the assistance of such a line because he has in mind the time values of the various forks.

However, instead of affixing precisely such a line for the purpose mentioned, I have compromised by dawing a line that would be of a little more clinical interpretative value. This line lies on a level a little lower than the longest vibration periods required to produce perception of sound in the almost totally deaf ear, and was obtained experimentally with the assistance of patients totally or almost totally deaf for the respective pitches. This is the borderline of total deafness. Of course, such a line is not and could not be mathematically determined. It is not necessary for the acumetric test, but is helpful in the interpretation. The acumetric line itself, relative to the normal, contains the record of acuity.

The borderline of total deafness has, however, the additional value of creating the conceptual effect of two fields: (1) the definite field of lost hearing (between the normal and the acumetric line), and (2) the approximate field of residual hearing (between the acumetric line and the borderline of total deafness).

SOME POINTS CONCERNING ROUTINE TESTS

In extremely hard of hearing patients who perceive the loudest intensities for only from two to four seconds, it will be sufficient to chart the number of seconds measured from the borderline of total deafness upward, thereby saving a great deal of time; for example, if one forcibly strikes a *c* 128 fork and the patient hears it for only four seconds, it should not be necessary to listen for the point of lost audibility in the examiner's ear. Count upward from the borderline of total deafness (which for *c* 128 is at forty-seven seconds) and place the mark at forty-three seconds. This fact in itself shows that the patient is almost totally deaf at this pitch.

In busy clinics and for the general run of cases, a test with the whole series of forks will probably not be practical. One may use two, three or four forks as may be desired in any given case. These may be sufficient, especially when the main object is to follow up the effects of treatment in catarrhal conditions or to note the results of inflation for diagnostic purposes. The effects of inflation are charted in interrupted lines.

The examining room must be very quiet, otherwise the margin of error may be sufficient to spoil the test. If the record is carefully made in the proper place, the margin of error is surprisingly small, as has been found by having records of the same patient made by two examiners.

After a little experience with this method the otologist will be rewarded with the satisfaction of establishing his tuning fork measurements visually, which will enable him and others to translate at a glance the diagnostic significance of the graph and, by comparative tests, the progress of the case.

I should be pleased to learn of the results obtained by others with this method. The charts are made for me by the Pfau Instrument Company, 2 East Twenty-Third Street, New York, and permission is given to reprint them.

570 Eastern Parkway.

ILLUMINATION OF THE EAR

A SHORT HISTORICAL SKETCH

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DETROIT

When the physician of today wants to examine an ear, he uses a concave head mirror and an artificial source of light, or an electric speculum. It is so very simple that it is almost hard to believe that this method has been developed step by step during a period of many years. Fabricius Hildanus[1] (1560-1634) devised a bivalvular ear speculum similar to the nasal speculum now in use. In Wilde's[2] textbook on the diseases of the ear, the following appears:

> Passing for the present the subjective symptoms, which will best appear in the words of the patient, let us inquire into the best method of forming a diagnosis from the physical signs presented. The patient being placed opposite strong, direct sunlight, with the head inclined at such an angle that the sun's rays may fall directly through a tubular speculum upon the membrana tympani, we first carefully observe the condition of the concha, external meatus, mastoid process, infrazygomatic region, and the space immediately below the lobe of the ear. The auricle, in its various folds, its color, its temperature in particular, its thickness as learned by feeling its hem or helix between the fingers, and the angle which it forms posteriorly with the cranium, together with the position, size, shape, and color of the external meatus, as seen without altering the relation of the parts, should be especially noted. The upper rim of the helix should then be grasped between the finger and thumb of one hand, and drawn upwards, backwards, and outwards, while the thumb of the other hand, placed in front of the tragus, by drawing it and the integuments forward upon the zygoma, exposes the outer third or more of the auditory canal to view. This little gorget-like instrument [depicted] will also be found useful in examining the external aperture. The finger should then be pressed deeply and firmly upon the movable root of the tragus, and backwards into the depression between it and the articulation of the jaw. While the finger is retained in this position the patient should be desired to open and shut the mouth, and the amount of pain or inconvenience experienced by pressure in those two different positions of the jaw accurately noted. The middle and forefingers should likewise be inserted deeply behind the ramus of the jaw towards the styloid process, and notice taken of the sensations there experienced. In 1827, Dr. Newburg [p. 70], in his "Memoirs et observations sur la perforation de la membrane du tympan," published at Brussels, recommended a slender horn tube,

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Submitted for publication, Nov. 12, 1931.

1. Hildanus, Fabricius, cited by Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, Ferdinand Enke, 1907.

2. Wilde, William R.: Practical Observations on Aural Surgery and the Nature and Treatment of Diseases of the Ear, Philadelphia, Blanchard & Lea, 1853, p. 56.

nearly four inches long, with a bell muzzle. This instrument, which is much too long, was the origin of the different varieties of tubular specula now in use. Subsequently Dr. Gruber, of Vienna, improved upon Newburg's idea, and to him I am indebted for the instrument of which I published an account in 1844.

An interesting contribution to the examination of the external auditory canal and of the drum membrane was published by von Troeltsch [3] in a number of consecutive issues of the *Deutsche Klinik*, beginning March 24, 1860. The following remarks are based on the articles by von Troeltsch, except the description of Erhard's apparatus.

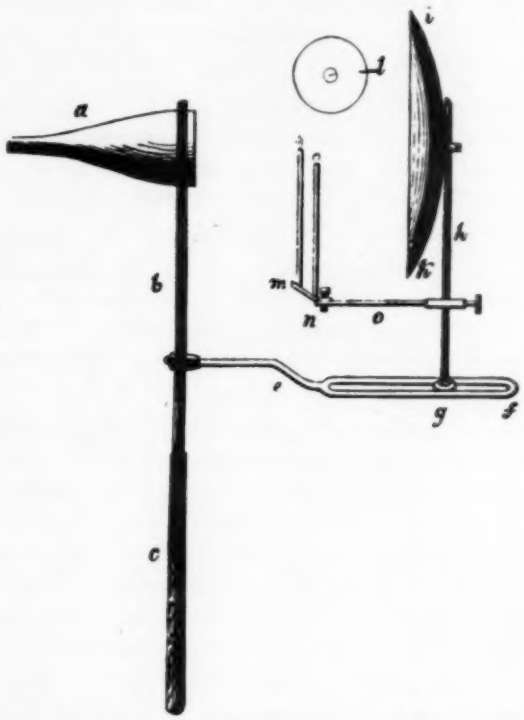

Fig. 1.—The ear illuminating apparatus of Julius Erhard.

The author mentioned the bivalve ear speculum of Fabricius Hildanus, also the ear speculums of Kramer and of Gruber. He mentioned that Gruber advised examination of the ear between 11:00 a. m. and 3:00 p. m., because, during that time, the light of the sun was at its best. Archibald Cleland used a convex lens of 3 inches in diameter and a candle opposite the middle of the lens in order to concentrate the light of the candle to illuminate the ear canal and drum membrane. Bozzini,

3. von Troeltsch, A.: Die Untersuchung des Gehörganges und Trommelfells, Deutsche Klin. **12**:113, 121, 131, 143 and 151, 1860.

six years later, in 1807, used a concave mirror behind candles. Toynbee in London, Triquet, in Paris and Erhard in Berlin used concave mirrors with a hole in the apparatus [4] (fig. 1).

In this instrument the silver speculum (*a*) is provided with an upper handle of steel and a lower of wood (*b, c*). One and one-half inches under the speculum, a penetrated handle made of steel (*e, f*) goes backward from the first handle. From the second handle, a third steel handle rises parallel to the first handle (*g, h*). This is armed with a concave mirror made of silver (*i, k*). This mirror can be moved in the window of the second handle (*e, f*). The diameter of the concave mirror is 2½ inches; the central opening (*l*) has a diameter of 3/16 of an inch, exactly as large as the narrow end of the speculum. It must be in the same plane as the narrow end of the speculum. A cross-stem (*o*) carries two wax candles which can be moved so that the light cones remain on the same plane as the hole of the mirror. The flames of the candle are on both sides of the hole of the mirror, but are not visible to the eye when it looks through the opening.

Buchanan, in Scotland, in 1825, used a box based on the idea of Bozzini. It was an instrument built on the principle of a telescope, which he named "inspector auris." Also, Jordan, of Manchester, and Warden devised instruments to examine the ear. Ménière used a candle in front of a spoon, the latter serving as a reflector. Voltolini, of Silesia, made use of a powerful light with a concave mirror behind it. (One learns that many endeavored to devise procedures that would facilitate the examination of the ear. It is therefore not surprising that similar thoughts occurred to various minds. To some degree at least, the ideas of several ran along similar paths.) In 1841, Hofmann, of Burgsteinfurt, described a device in Casper's *Wochenschrift*.[5] He removed the material in the center of the reverse side of an ordinary mirror, used for shaving, and looked through the clear glass. Rau,[6] in his textbook, mentioned Dr. Alther of St. Gallen, who used a perforated metal concave mirror. Von Troeltsch demonstrated his concave mirror in the Society of German Physicians in Paris, in the winter of 1855-1856, and in 1858 in Wuerzburg. The diameter was 6 inches, the focus 4 inches and the diameter of the central opening was 2¼ lines. (A line is a tenth of an inch.) Von Troeltsch stated that a diameter of from 2½ to 3 inches was sufficient. Edward Jaeger [7]

4. Erhard, Julius: Rational Otology, Erlangen, Ferdinand Enke, 1859, p. 82.

5. Hofmann: Beitrag zur Untersuchung des äussern Gehörganges, Wchnschr. f. d. ges. Heilk. **7**:10, 1841; cited by von Troeltsch.

6. Rau, W.: Lehrbuch der Ohrenheilkunde für Aerzte und Studierende, Berlin, H. Peters, 1856; cited by von Troeltsch.

7. Jaeger, E.: Ueber Staar und Staaroperationen, Vienna, L. W. Seidel, 1854, p. 84; quoted by von Troeltsch.

stated that his eye mirror could be used for examination of the outer ear, if a concave mirror with a focus of 4 inches was inserted.

Czermak,[8] in his article on the laryngeal mirror, said:

> If we approximate an intense flame to the open mouth of a patient, the observer can examine the pharynx and employ the laryngoscope, in placing his eye very close to the flame; and this latter may be masked by a screen or a reflector, as in the simple form of the ophthalmoscope of Helmholtz. In order to have both hands free, I have placed a candle and its reflector before the eyes, by means of a handle fixed between the teeth. But I have succeeded in obtaining a result more complete and easy still in using the large ophthalmoscope of Ruete, joined to a powerful moderator-lamp. Since then I have used a whiter and more intense light, such as, for example, that of ordinary gas, camphine, etc. Besides, natural philosophy furnished us with sufficient means for increasing the intensity of artificial light. On introducing the employment of the ophthalmoscope in laryngoscopy, I have accomplished the necessary concentration of the rays of artificial light, in a manner by which these researches have become independent of the light of the sun. But I have found it very convenient, on the other hand, always to use for the purposes of light, reflecting mirrors pierced by a hole in their center, even when we have the light of the sun at our disposal; in that case, the concave mirror is perhaps replaced by one that is flat. The reasons which have determined me to employ the perforated mirrors under all circumstances are the following: 1. The eye of the observer can always see with facility by looking through the opening in the direction of the rays reflected by the illuminating mirror. In this way, the same objects that are found reflected by the laryngeal mirror are always observed illuminated in the most vivid manner. 2. There is never any risk of throwing a shadow with the head upon the objects which it is desired to illuminate. 3. Lastly, the individual can be examined in almost any position that is desired. The illuminating mirror, which is flat or concave, possesses a circular form, a diameter of three to four inches, and a focal distance of eight to twelve inches; it is attached to a stem with two branches, and remains movable upon its horizontal axis; it can be arranged at a convenient position before the eye; it is there maintained either by the aid of the hand which holds the stem, or, to permit of both hands being free, by the aid of a handle introduced between the teeth, or, again, by means of the frontal bandage of Kramer, which I have applied to this use, or, lastly, according to the custom of Semeleder, which I have, and Stellwag, by arranging it in a similar manner to that of a pair of spectacles.

Politzer,[9] in the third edition of his textbook, spoke of a head mirror of from 7 to 10 cm. in diameter with a focus of from 12 to 15 cm., but did not mention the size of the central opening. Also, in the fifth edition of the same text,[10] he does not say anything about the size of the central opening. During my stay as intern in the Massachusetts Charitable Eye and Ear Infirmary in Boston, 1896-1897, I

8. Czermak, J. N.: On the Laryngeal Mirror, Gaz. hebd. de Vienne, no. 13, March 27, 1858; also in Selected Monographs on the Practical Use of the Laryngoscope, London, New Sydenham Society, 1861.

9. Politzer, A.: Lehrbuch der Ohrenheilkunde für practische Aerzte und Studierende, ed. 3, Stuttgart, Ferdinand Enke, 1893.

10. Politzer (footnote 9, ed. 5, p. 82).

introduced a larger central opening of the head mirror.[11] Musehold, of Berlin, had previously used a large-holed reflector for photography of the larynx and, according to a private communication (from H. Pfau, Feb. 16, 1911), had used the large-holed head mirror in his practice.

The Vienna school used head mirrors with somewhat large oval openings in the center based on the principle that the visual field is better served by this arrangement. Dr. Benesi, of Vienna, removed a segment of the reflector vertically, so that the nose does not touch the mirror. This device requires two knobs, so that the mirror can be used also in front of the other eye. Dr. Trautmann, of Munich,[12] devised a mirror without a central opening. On the periphery of the mirror, a semicircular segment of about one-third diameter of that of the mirror is removed. Hasslauer stated that binocular vision is made possible

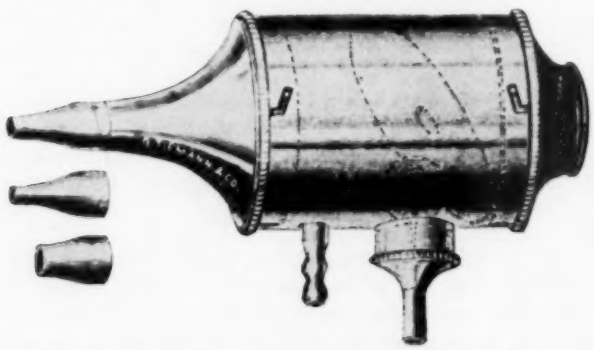

Fig. 2.—The electric ear speculum of the author.

without removal of the mirror from the eye, "which is of incalculable benefit when working with sterile hands."

The various electric headlights can be divided into three classes: (1) those without concentration of the light rays; (2) those with concentration of the light by a concave mirror, Klaar's headlight, with two holes in the mirror, one for each eye, and (3) those equipped with a lens in a movable hood (L. Kuttner, Williger, Kirstein-Killian, etc.). The two latter classes are more useful, in my opinion, because a strong light can be focused in the depth. The first class is more adapted to work in the throat.

For a number of years, electrically lighted appliances, which carry the light within the speculum, have come into use. Such an ear speculum,

11. Amberg, E.: Tr. Michigan State M. Soc., 1899; J. Michigan M. Soc. **30**:785 (Oct.) 1931.

12. Trautmann, cited by Hasslauer, W.: Die Ohrenheilkunde des praktischen Aerzte, Munich, J. E. Lehmann, 1911.

so far as I know the first one, was described by me in the *New York Medical Journal*[13] in 1899 (fig. 2).

There is a small electric light bulb within the speculum. (Burnett,[14] of Philadelphia, in 1899 described a modified Siegle's pneumatic aural speculum which does not contain the light within the speculum.) Other electrically lighted ear speculums have come into use. Some of them are rather cumbersome, if the source of light is contained in a storage battery which is connected directly with the speculum. Only one firm, I believe, has detached the source of light from the ear speculum. This makes the speculum lighter and its handling easier, and it is more agreeable to the patient and to the physician. I think that the shortening or removal of the handle may make the speculum still more useful.

Magnification of the parts observed can be obtained in several ways. A lens can be attached to the reflector or the lens can be held in the hand, or it can be placed in the electric ear speculum. Luescher,[15] in 1927, devised an ingenious apparatus which magnifies from ten to fifty times. The source of light is a lamp with a spiral thread of 8 volts and 33 candle-power. The light is red-free.

Further progress in the illumination of the ear might be made by applying the principle of the endoscope for visual examination of the epitympanic cavity and surrounding parts, under favorable circumstances, namely, if a sufficiently large perforation exists, or if a destruction is present. Such a tympanoscope may prove to be useful in some instances and may replace the small mirrors devised for this purpose, provided that it is technically possible to make such an instrument.

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13. Amberg, E.: New York M. J. **69**:1725 (May 20) 1899.

14. Burnett, C. A.: A Modified Siegle's Pneumatic Aural Speculum, J. A. M. A. **32**:1212 (June 3) 1899.

15. Luescher, E.: Microscope for the Ear, Magnifying from Ten to Fifty Times, Ztschr. f. Hals- Nasen.- u. Ohrenh. **17**:403 (Feb. 22) 1927.

INTRATRACHEAL INHALATION ANESTHESIA IN PRACTICE

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NEW YORK

In previous communications,[1] the technic of inhalation anesthesia described suggested the following considerations:

It was stated by Dr. Chevalier Jackson that intubation by the method advocated was nontraumatic, Dr. Jackson's opinion being, "There is no reaction of any consequence in an operation as long as two hours. There is vastly more reaction from the bubbling back and forth of ether laden mucous when ether is given by the open-drop method."

The disadvantages of direct laryngoscopy and the presence of the tube were believed to be counterbalanced by the simplicity of the equipment providing an operative field free from escaping anesthetics, an uninterrupted normal respiration, conservation of carbon dioxide, exclusion of foreign matter, such as blood, vomitus, mucus, etc., complete control of a lightly anesthetized patient, functioning facilities for artificial respiration, protection of the trachea from external pressure, an aseptic operative field and a means whereby any anesthetic vapor could be conveniently and safely administered to the patient.

Rubber and composition tubes were discarded in favor of a modified bronchoscope, the average margin of possible trauma of which had already been measured by extensive and constant usage in the hands of many bronchoscopists.

To the comparatively safe and nontraumatic technic of intubation as practiced in ordinary bronchoscopy on the conscious patient is added the control of the reflexes and the muscular relaxation that accompanies general anesthesia. The resultant ease of exposure and absence of spasm place the technic within the range of almost any one who wishes to avail himself of its benefits.

The following matter is a candid description of my actual experience with intratracheal anesthesia during the last four years, this method having become the method of choice in all operations about the head and neck. As at least 95 per cent of the intubations have been done in so-called private cases, it will be understood that the maximum personal obligation has accomplished this choice of method.

Submitted for publication, Dec. 30, 1931.

1. Flagg, P. J.: Intratracheal Inhalation, Arch. Otolaryng. **5**:394 (May) 1927; Exposure and Illumination of Pharynx and Larynx by the General Practitioner, ibid. **8**:716 (Dec.) 1928.

The number of operators, the variety of their technic and the number of hospitals in which these operations took place have been comparatively large, and the opportunity for routine team work correspondingly small.

Careful perusal of the interesting facts that have appeared in relation to the following points will preserve the beginner from occasional embarrassment. The points to be discussed are: technic of laryngoscopy, preliminary medication, the protection and the care of the tubes, intratracheal suction, pharyngeal packs, the cough spasm, respiratory failure, intubation and cocainization of the larynx, extubation, anesthetic agent, intubation in pulmonary tuberculosis, definite and borderline indications for intubation, routine laryngoscopy and the reaction of the patient, the surgeon and the anesthetist.

VARIATIONS IN TECHNIC OF LARYNGOSCOPY

It has been found that entirely satisfactory exposure of the larynx may be obtained in the average case when the patient lies flat on his back with the head on a level with the shoulders. The actual exposure may be said to take place as a result of a bimanual manipulation, beginning with a partial extension of the head brought about by pressure on the forehead by the right hand. The laryngoscope, which is grasped in the left hand, is placed between the teeth and against the dorsum of the tongue. The free edge of the epiglottis is readily brought into view; the lip of the laryngoscope is then depressed so that it will lift the epiglottis and the dorsum of the tongue to which it is attached. While this exposure is being made with a lifting rather than a prying effort, extension of the head is continued with the right hand. The posterior commissure and, gradually, the entire larynx then fall into view. Experience will enable one to slip the laryngoscope away from the incisors over the bicuspids or the molars; the degree of exposure and the rapidity with which this may be made depend very largely on the presence or the absence of the upper teeth and their condition of repair. One must beware, however, of the easy approach of the partially anesthetized edentulous patient, who will develop a sharp spasm on intubation because of the low degree of anesthesia which permitted the original exposure. One becomes quite adept in sizing up the difficulties presented by a patient on whom anesthetization and laryngoscopy are about to be done. As a general rule, the larger the face and the features, the easier and the more satisfactory the intubation. Small, high-strung, nervous patients will sometimes give a surprising amount of difficulty until they are thoroughly controlled by the anesthetic. Because of the frequent change in operators, place of operation and the type of patients under treatment, I have hesitated to attempt routine intubation with anesthetic agents that do not bring about relaxation, such as nitrous oxide and oxygen or ethylene. The routine anesthetic

is ether, because of its wide margin of safety and its measurable degree of relaxation. Chloroform is available for cases of respiratory obstruction from tumors, abscesses or similar conditions occurring in the airway. It is possible, occasionally, to perform laryngoscopy on a patient by the use of avertin in the 100 mg. dosage. However, it has been found that laryngeal reflexes are usually very active in patients who have received only avertin. After intubation with the routine gas-oxygen-ether induction, any anesthetic agent desired may be employed. In cases of diathermy, or when the actual cautery is used in the nose and throat, ether is discontinued and chloroform vapor substituted. Intratracheal inhalation offers the safest possible technic for the administration of chloroform, for the reason that the intratracheal tube does away with the possibility of respiratory spasm and a patent airway is constantly present. In conditions in which it is desirable to maintain anesthesia by gas-oxygen, this anesthetic may readily be given by any available gas machine. The tendency to increase bleeding and the increased respiratory excursion that accompanies gas-oxygen has favored the retention of the simple technic of ether inhalation. When it is recalled that there is no objection to the anesthesia being so light that the patient occasionally strains, that even if he does, he cannot pass out of the control of the anesthetist, as is the case in any other technic, the small amount of ether required to maintain such anesthesia offers little difficulty. In a previous paper, it was pointed out that the rigid section of the intratracheal tube was of a definite length, in order that it might be self-retaining when the patient's head was in a straight or flexed position. From this consideration it becomes apparent that the return of the reflexes and muscular rigidity accompanying a low level of maintenance may offer resistance to the removal of the tube. This resistance is very real and is best overcome by a few respirations of concentrated ether vapor. Therefore, because of the necessity of relaxation for intubation and for extubation and because of the difficulty of judging color in the darkened room used for operations on the nose and throat, ether has been the routine anesthetic agent, administered by the simple tin can method previously described.

In order to strengthen the laryngoscope that has been used, a new model has been developed in which the handle and the blade are one, as shown in the illustration (fig. 1); the handle contains the batteries, and the electrical connections have been brought down to the irreducible minimum; the blade of the laryngoscope has been increased in length in order to facilitate the exposure of the occasional deep larynx that one meets in practice. The laryngoscopes are so made that the blade and the lamp may be sterilized by boiling. In practice, I use alcohol or ether for sterilization. The lamps supplied are sturdy and will stand a good deal of usage. Occasionally, however, a lamp will

fail. Lamps should invariably be tested, in place, in the laryngoscope before anesthesia is induced. Trouble with the light may occur in three locations: batteries, wiring connection and lamps. Batteries seldom fail completely; there is a glow even from a very weak battery. Wiring connections almost never fail. Lamp contacts are the most frequent source of lighting difficulties. This difficulty will usually be found to be due to corrosion at the point of contact, or to a lamp that cannot be screwed far enough into the socket, owing to accumulations of corrosions about the rim. Lamps occasionally burn out. In this case, the lamp glass will be found to be darkened in color. Because of the possibility of trouble with the light, it is desirable to carry extra lamps or duplicate blades.

Fig. 1.—Flagg's one piece laryngoscope.

PRELIMINARY MEDICATION

In my previous communication, I advocated the routine use of morphine and atropine forty-five minutes before operation. I found this view to be fallacious, for after a long series of cases with and without medication, I found that the patients in whom medication was omitted offered a much easier laryngoscopic exposure. I believe the explanation to be as follows: Preliminary morphine depresses the respiration; ether is a respiratory depressant after the period of excitement and rigidity. With preliminary medication, a condition frequently develops in which muscular rigidity still persists in the face of a shallow, rapid, ineffective respiration. The margin of safety between good relaxation and the disappearance of the respiration has been reduced to a dangerous point; on the other hand, a patient who has not had preliminary medication will arrive at a point of complete relaxation long before the respiration becomes depressed, offering a wide margin of

safety for the patient. After intubation, on the other hand, medication may be freely used and will serve to reduce the amount of general anesthetic necessary to maintain the patient quietly. In long operations, such as combined nasal and throat operations, pansinus operations, mastoiditis, etc., this technic is frequently employed.

Avertin may be used as a basal anesthetic. It is felt that because of the protection to the respiration afforded by the patent inhalation tube, avertin is robbed of its gravest danger, namely, respiratory obstruction.

INHALATION TUBE

All inhalation tubes are now chrome-plated. The wire spring is made of noncorroding metal. Once or twice when a tube had been repaired by the replacement of a steel spring, the latter separated while in use, causing embarrassment; the separated sections, however, were held together by the friction of the Penrose rubber covering. When a patient is to be maintained with a very light anesthesia, a mouth gag should remain in place, otherwise a spasm may induce him to bite into the tube, throwing this out of line and requiring replacement. It is customary to install a mouth gag before laryngoscopy and intubation, as the spasm sometimes induced by the intubation of the tube may cause the patient to close his teeth. The more frequently a tube is used, the less frequently is it necessary to change the rubber covering. A tube that is used only occasionally becomes dried out, the rubber adhering closely to the metal and giving a great deal of difficulty in removal; such adherent rubber may be removed by scrubbing with sapolio with the opturator in place. The rubber covering should always be removed against the closed spring, i. e., from the hose connection to the tip. A spring that has been extended too vigorously will set and become useless. New sections of rubber are readily applied by moistening the tube with a little green soap, wetting the inside of the new rubber tubing and slipping it over the tip and the shaft of the tube. Care must be taken to rinse the tube out carefully before use or the rubber section may become displaced. Sticky rubber covers should immediately be removed and discarded. Covers with holes or torn sections should be removed.

For the last two years, it has been my practice to lubricate the tip of the intubation tube before introduction; this may be done with petrolatum. There seem to be no reasonable objection and much protection from this technic.

SUCTION

Adequate suction is essential to this technic. Suction must not only be available but must be functioning at the instant it is needed. It should be made during induction of the anesthesia; the elimination of morphine and atropine exposes the patient to the salivation common to

the inhalation of ether. This saliva and mucus should be removed completely before intubation; after intubation, the catheter used for the purpose should be slipped into the intratracheal tube and intratracheal suction practiced. Quiet respiration will guarantee the absence of tracheal mucus. It is extremely important that an open-end catheter of the correct size, 14 French, be employed. A large catheter will block the intratracheal tube, and during the inspiration of the patient, saliva or blood which may be in the pharynx will be sucked in around the sides of the tube. Before extubation, careful pharyngeal suction in both the nasopharynx and the hypopharynx should be employed. It is an excellent practice to allow the patient to recover his vomiting reflex while he is still on the table and under the protection of adequate suction. When there is a doubt as to the quality of the suction available, the anesthetist should provide his own.

PHARYNGEAL PACK

In the early use of the intratracheal method, packing the pharynx was practiced in all cases, tonsillectomies included. It was soon found, however, that in the case of tonsillectomies, even a small pharyngeal pack was in the way. Furthermore, it was not essential because pharyngeal suction can be applied in these cases to cover the accumulation of excessive fluid. The ordinary pack for nasal operations consists of a 2 inch gauze strip from 12 to 20 inches long, depending on the capacity of the pharynx. When induction is complete, the mouth gag is placed, the pharynx aspirated and the intratracheal tube introduced; the gauze is then introduced into the pharynx as deeply as possible, being directed over the surface of the tongue by the index finger of the right hand. Care must be taken to observe that the pack does not include the tongue, causing occasional trauma to the frenum. It has been observed in a number of cases that this packing technic occasionally causes soreness of the mucosa of the roof of the mouth and pharynx. This does not occur when saliva is present and may be quite effectively controlled by lubricating the parts with petrolatum, or by moistening the gauze with petrolatum.

I have been reluctant to use the so-called subglottic pack by means of a small balloon attached to the intratracheal tube. While I employ this procedure in resuscitation, I feel that the normal resistance presented by the technic of exposure, the occasional active laryngeal reflex and the fact that any rubber air-container cannot be depended on when it is most needed places this technic outside the range of normal practicability. It is difficult enough to intubate as a routine measure by smooth cylindric tubes without complicating the procedure by an increased diameter and a rough surface.

THE COUGH SPASM

Early in intubation, one was occasionally disturbed and alarmed to find that the patient, immediately following intubation, entered a period of prolonged apnea, accompanied by cyanosis of increasing intensity. After what seemed to be an interminable time, respiration began again, rapidly becoming normal, and the color cleared completely. With a view of obtaining light on this phenomenon, an inquiry was sent to Dr. Jackson. His comments are as follow:

> The cyanosis that you have noticed is probably due to the cough reflex; but you do not get the cough because the cords are separated in the position of complete abduction. The best remedy for this condition is to get the patient completely under before you introduce the tube. There is no danger whatever from the laryngeal reflex in your hands. There is no need for any nerve block or any of the other things that used to be considered necessary. You can introduce a tube into the trachea, and with the trachea intubated, there is no danger whatever, so far as the airway is concerned, nor, so far as any reflexes are concerned.

This communication is dated Jan. 31, 1928. Since that time, the cough reflex spasm has been seen in perhaps one in ten cases, the intensity of the spasm and its duration being directly in proportion to the degree of anesthesia. I find myself frequently betrayed into an early intubation in the case of little children and in edentulous adults in whom laryngoscopy is so easy that I do not have to bring about the anesthesia necessary in the average case. In addition to the infrequent appearance of the spasm due to more deliberate induction, it can practically be controlled by either blowing directly into the tube, thereby stimulating the Herring-Breuer reflex, or by blowing a little oxygen immediately into the tube, which oxygen is immediately absorbed, clearing the cyanosis and reducing the duration of the spasm. Occasionally, when the anesthetic has become light during intubation, a spasm is induced by the first inhalation of concentrated ether vapor. This is immediately remedied by reducing the ether concentration or removing the anesthetic until respirations are resumed. Continued cough may follow the contact of the tube with the carina, in the event of a tube being introduced beyond the necessary depth. It is to be observed that the larynx rapidly develops a tolerance for the presence of the tube, and the occasional straining or cough that may result from a continued light anesthesia is of short duration, of little consequence and easily controlled. The assumption that an indwelling intratracheal tube is a continued source of irritation to the anesthetized patient is not borne out in practice. After the actual intubation, it is desirable to retain the laryngeal tone, as a completely relaxed larynx permits foreign matter, for example, blood and saliva, to find its way into the trachea along the sides of the tube. In practice, when the cough reflex is now encoun-

tered, it is treated as a matter of course, with the confidence that it is more distressing to observe than dangerous.

EMERGENCY TREATMENT IN RESPIRATORY FAILURE

There is no doubt that the instrumentarium provided by intratracheal inhalation anesthesia affords the simplest, most direct and most efficient means of meeting respiratory failure. Resuscitation by mouth to mouth insufflation was reported.[2] In addition to the actual resuscitation of a patient, the increased margin of safety presented by this technic with ether as the anesthetic agent gives a margin of safety to the patient that is so wide that the usual anxiety normally present in anesthesia of the head and neck is completely disposed of. Once intubation has been completed, I feel that my troubles and anesthetic dangers are over for the case in question, for variations in the position of the tube and intratracheal suction are routine procedures that give rise to no disturbance.

POSITION OF THE INTRATRACHEAL TUBE DURING ANESTHESIA

For the reason that the tube changes its position when the patient's head is extended, flexed or rotated, it has seemed wise to maintain a constant diameter throughout. Variations in diameter, such as are exhibited by the O'Dwyer tube, cause variation in pressure against the cords, and consequently, a stirring up of the laryngeal reflex. Shortly after intubation, the larynx seems to acquire a tolerance for the tube. This tolerance is disturbed only by sudden movements of the head by the operator. The rigid portion of the tube, the so-called intratracheal section, is of such a length that when intubated to its full length the tip will miss the bifurcation and the carina by at least 1 inch. The large adult tube has been made of such a length in its rigid section that it is self-retaining; that is, it will not slip out of the glottis when the head is flexed. While the length of the rigid section makes extubation more difficult, the self-retaining quality of this feature has favored its use. When the large tube is intubated in the comparatively large larynx of the young patient, the distance between the vocal cords and the carina is naturally less than in the full-grown, long-necked adult. Therefore, care should be taken that the tube does not find its way into one bronchus, thereby reducing the tidal air one-half. The best test of the correct position of the tube is that of free and full respiration through it. While this may be measured by a spirometer, in practice, the sound of the air passing through the tube is noted. In introducing

2. Flagg, P. J.: Successful Mouth to Mouth Insufflation in the Asphyxiated Adult, Am. J. Surg. **8**:1204, 1930.

the tube, all that is necessary is to pass the cords; flexion of the head following this will cause the tube to find its proper level.

COCAINIZATION

The application of 10 per cent cocaine to the exposed cords produces prompt and complete relaxation, accompanied by a wide open glottis. While useful and necessary in bronchoscopy in adults and in operative work under general anesthesia, it has seemed unwise to employ this technic as a routine measure in intratracheal inhalation anesthesia. While intubation is facilitated, the loss of tone of the cords is likely to persist for some time and to defeat the object of intubation, namely, permitting the passage of foreign matter into the glottis. When anesthesia is induced with gas, oxygen and ethylene without ether, the possible trauma to the active larynx on intubation may be more serious than the duration of the relaxation produced by cocaine. In this event, and when it is important to intubate with the minimum amount of general anesthesia, cocainization may be practiced.

Cocainization in children is not done owing to the danger of absorption.

EXTUBATION

Before extubation, a mouth gag should be set in place, and it should be the unvarying rule to practice careful aspiration of the hypopharynx and upper part of the esophagus, with particular reference also to the nasopharynx, for one will almost always find accumulated blood clots or foreign matter high up above the soft palate when the pharyngeal pack is removed. After aspiration has been completely performed, the head should be extended and traction made on the tube. In doing so, the fingers of the right hand should slip into the pharynx around the tube, reaching for the upper end of the rigid section. This should then be lifted upward and outward between the teeth. If the patient has been carried under a very light anesthesia, traction on the tubes will immediately stir up laryngeal reflex, and the tube will be gripped firmly by the cords. Extubation should not be attempted under these conditions. A few breaths of ether vapor will cause temporary relaxation, which will permit of easy extubation. Immediately following extubation, aspiration should be again performed and the patient assisted in recovering his ordinary respiratory rhythm by extending the head and lower jaw. Following extubation, a short period of apnea may follow which will promptly pass off. If the patient is relaxed during extubation and if the vocal cords are sluggish from deep anesthesia, the airway should be guarded by suction until the reflexes return; this will usually occur in a few minutes.

AGENTS EMPLOYED

Ether, chloroform, gas-oxygen or ethylene may be used in intratracheal inhalation. For practical purposes, the anesthetic agents are gas-oxygen, ether and chloroform. Except in the case of very small children, gas-oxygen is used for induction, and ether is then resorted to. My experience with ether as an agent to maintain anesthesia has been so satisfactory that I abandon its use only under special indications, as, for example, in the event of a patient with an acute pulmonary condition, glycemia or acute nephritis. I like ether to maintain intratracheal anesthesia because of the quiet, respiratory effort, the full oxygenation and, consequently, the reduced bleeding that accompanies it. Ether administered in this fashion will maintain a quiet anesthesia on a level that is so light that the patient's eyes will move about, and other indications of light anesthesia will be evident, such as occasional movements of the head and attempts to cough. Vomiting may also occur, and it may be remarked that this is one form of anesthesia in which vomiting is of no consequence, for, when the pharyngeal pack is in place, vomited material remains in the esophagus. Even when there is no pack, the laryngeal reflexes are proportionally active, the cords close about the tube, and the patient breathes freely and comfortably through a pharynx that may be filled with vomited material. This foreign matter, which may be removed at leisure, does not interrupt the course of the anesthesia. When gas-oxygen is resorted to to maintain anesthesia, a hypodermic injection of morphine may be administered after intubation. This will reduce the patient's tendency to hypernea and will increase the limits of oxygen toleration. When gas-oxygen is used to maintain anesthesia, it is well to resort to a few breaths of ether on extubation, except in the case of patients with a very large pharynx. Any source of gas and oxygen from any make of machine will provide a satisfactory gax-oxygen anesthesia. The illustration (fig. 2) shows a simple assembly attached to the intratracheal tube which I have found useful in maintaining anesthesia with gas-oxygen. In diathermy, or in the use of the actual cautery in the mouth, I have used chloroform to maintain anesthesia and occasionally to induce it. At first it seemed best to blow chloroform vapor into the inhalation tube by means of the ordinary Juncker bottle. This technic was later simplified by pouring a little chloroform into the ether can and allowing the patient to breathe of this, as required. Because of the impossibility of the laryngeal spasm, shutting off the respiration and inducing subsequently a deep inspiration, I have felt that this method is probably the safest one by which to administer chloroform.

The rubber inhalation tube is, of course, never attached to the can containing the chloroform, this tube being held some distance from it

or in contact with it, according to the depth of the anesthesia required. It is entirely practical to shift from ether to chloroform and back again to ether as the operative requirements and use of the diathermy indicate.

It has been possible to perform intubation on an edentulous patient under the basal effects of avertin. It has been possible to perform laryngoscopy on others. As a rule, however, the addition of ether is essential to complete the induction in these cases.

I believe that it is the duty of all those who use avertin to provide themselves with the technic and the facilities for intubation. By this means alone, the relative safety of avertin may be greatly increased.

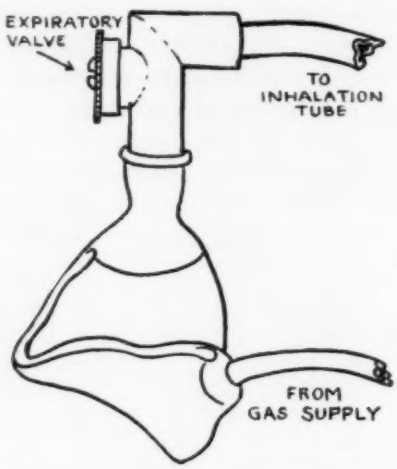

Fig. 2.—Assembly for intratracheal gas-oxygen or ethylene.

INTUBATION IN TUBERCULOSIS

The question has been raised, does intubation such as is practiced in intratracheal inhalation anesthesia predispose a tuberculous patient to laryngeal tuberculosis? This question has both a theoretical and a clinical aspect. The question may be best answered by inquiring into the degree of trauma accompanying intubation and determining whether such trauma may actually provide a focus of tuberculous infection. It has been argued that the presence of the intratracheal tube produces a condition of continuous spasm which may result in trauma. Experience indicates that whatever spasm may occur on intubation is brief in duration and does not persist. The question of trauma by the intratracheal tube is one that may be directly and immediately answered by recalling that the tube used is a typical bronchoscope in its size and shape, and that ordinary bronchoscopy does not traumatize the cords.

When one adds to this general fact the important consideration that routine intubation is done under the relaxation of general anesthesia, the reasons for traumatizing the cords are certainly reduced to a negligible factor and should not offer any more difficulty than the routine treatment of any mucous membrane in any nose and throat procedure. With a view of determining Dr. Jackson's reaction to this question, an inquiry was made, eliciting the following reply (Nov. 11, 1931):

> On theoretical grounds only, one might state that there might be a possibility of favoring a laryngeal tuberculosis in a tuberculous patient by the passing of a bronchoscopic tube, if trauma were inflicted, but I see no reason why there should be any trauma to the larynx from the insertion of a bronchoscope. Personally, I do not believe that laryngeal tuberculosis is an infection through the mucosa. I think it is through the lymph channels, but this is an opinion, not a proved fact. The foregoing is strictly theoretical.
>
> As a clinical fact, I have never seen any case, in which I saw reason to suspect that a laryngeal tuberculosis had followed a bronchoscopy on a patient with pulmonary tuberculosis. If it came to a bronchoscopy on myself or any of my family, if the patient were tubercular, I would not hesitate to sanction a bronchoscopy on the basis of the purely theoretical considerations mentioned.

In the course of the last four years, my experience bears out the contention that routine intubation may be practiced without trauma. Over this period only one case is recalled in which an abrasion of the cords by a dry tube in a dry larynx became secondarily infected and resulted in hoarseness. Occasionally a mild cough developed lasting for two or three days.

On the other hand, it is generally agreed that the postoperative reaction in cases of pulmonary tuberculosis is due, not to the irritating effects of the anesthetic, or as the effect of instrumentation, but as a direct result of the inspiration of the contents of tuberculous cavities into noninfected portions of the lung. It is also maintained, and has been emphatically pointed out by Pol Coryllos, that the use of intratracheal anesthesia, because of the facilities for intratracheal and bronchial suction which it offers, is the ideal means of maintaining anesthesia in such cases. Furthermore, it is also generally granted that the effects of ether *per se* do not cause a lighting up of tuberculous foci except indirectly, by promoting excitement, cough and emptying of tuberculous cavities combined with the aspiration of saliva which may have been allowed to accumulate in the pharynx. When saliva is aspirated and when intratracheal suction is practiced, the postoperative recovery is prompt and complete. Further studies relating to anesthesia in tuberculosis are now in progress.

SPECIAL CONDITIONS IN WHICH METHOD IS INDICATED

I believe that intratracheal inhalation anesthesia is positively indicated as a method of choice in nasal surgery, i. e., operations for pansinusitis, for conditions of the intranasal antrum and for tumors of the nasal pharynx. It is extremely valuable in all forms of oral surgery; in tonsillectomies on adults when the surgeon finds it practical to accommodate his technic to the presence of the intratracheal tube; in all forms of pharyngeal and laryngeal obstruction, either internal or external; in operations on the eyes of children for recession, advancement and other conditions; in prolonged operations about the ear in large vigorous subjects, and in all operations about the face, such as nasal reconstruction and the large variety of plastic operations. It is also positively indicated in cases of intestinal obstruction in which there is fecal vomiting, or in cases in which operations must be done shortly after the patient has had a meal. The method should be available for use as a means of resuscitation wherever the basal anesthetics are used, such as avertin, sodium amytal, etc., and in all desperate cases in which immediate freedom of the airway may suddenly be called for.

ROUTINE LARYNGOSCOPY IN ANESTHETIZED PATIENTS

The technic of intratracheal inhalation anesthesia turns on a single factor, the ease and the rapidity with which the larynx may be exposed. With a view of making such facility practical, it is earnestly advocated that in all patients experiencing nose and throat operations, children as well as adults, laryngoscopy be done at the conclusion of the operation. This routine laryngoscopy serves two distinct purposes: First, it enables the operator to become intimately acquainted with the technic required for intubation; second, it allows an inspection of the hypopharynx and the removal of blood clots that are almost always present at the conclusion of an operation on the nose and throat. It is only by practice under these conditions that the operator may acquire such familiarity with exposure that he may perform laryngoscopy quickly and without trauma as the patients appear before him for intubation. Expertness in this technic will also be a life-saving factor in patients who require immediate intubation for artificial respiration.

REACTION OF THE PATIENT TO INTUBATION

I have made an honest effort to obtain the postoperative reaction of patients on whom I have done intubation. Postoperative reaction, as experienced by the patient, has been conspicuous by its absence. An increasing number of physicians and nurses on whom intubation was done with their knowledge of the procedure have failed to experience

postoperative discomfort. I frequently speak with the patient within an hour after operation, and even at this proximity to extubation, I have discovered no ill effects. When a patient has been carried with a light anesthesia and extubation done when the reflexes are active, some stridor has immediately been observed, passing into a mild degree of hoarseness which gradually disappears. In one case of a child 2 years old, this condition following extubation in the face of spasm was quite marked. The reaction disappeared, however, within a few hours. A number of patients have complained of sore throat, but on investigation it was found that this pain was referred from the operative site and on disappearance would recur when the operative wound was treated. Several patients have complained of enamel chipped from the incisor margin, and sometimes from damaged dentures. Porcelain fillings are extremely brittle and are damaged by slight pressure. This difficulty has been reduced by using a mouth gag, by getting sufficient relaxation and by exposing from a bicuspid or a molar site. It is well to protect the gums of edentulous persons during laryngoscopy. No case of glottic edema of even a mild degree has been observed, although edema of the arytenoids has occasionally been seen before intubation. The outstanding characteristic of the technic appears to be the tolerance of the glottis to the tube employed. As more than 90 per cent of the intubations have been done in private practice, the element of personal reaction has been rather thoroughly covered.

SURGICAL REACTION

The reaction of the surgeon to the technic has varied. The resistance found appears to be chiefly due to unfamiliarity with the technic and to the routine reaction of the patient. The method has been almost universally welcome in all nasal operations and in difficult cases of oral surgery. In tonsillectomies on adults, it is usually necessary to use the method in order to experience the advantages of a quiet field from which the element of respiration has been removed. The position of the intratracheal catheter in the mouth threatens to embarrass the operative field. The advantage, however, of being able to dislocate the tongue and place it in any position without regard to the patient's respiration usually overcomes the embarrassment of the tube. Most operators, after using the technic a number of times, learn to disregard the tube altogether. An important factor in tonsillectomies is proper retraction for adequate exposure. The average adult is more satisfactorily maintained in anesthesia by this technic than by any method with which I am familiar. It is a method of choice in all cases. Occasionally, however, an obese person, with a small mouth, deep pharynx and large tongue, presents considerable difficulty. These cases require cooperation

on the part of the surgeon if the technic is to be used. When it is found that the presence of the tube is embarrassing to the surgeon, extubation is easily done without disturbing the level of the anesthesia, vapor methods then being resorted to. These are borderline cases and must be treated individually. Intubation need not be done for tonsillectomy on little children who may be maintained on such a light anesthesia that the cough reflex prevents aspiration. However, it has been frequently observed in little children that when profuse salivation occurs during induction, with partial aspiration of this saliva and accompanying asphyxia, proper relief can be secured and maintained only by intubation and intratracheal suction. The immediate oxygenation in these cases occurs with astonishing rapidity. In work on the eye in children, intubation is a method of choice and by far the safest technic that may be employed. Owing to the presence of the tube in the operative field, it is desirable that the surgeon familiarize himself with the technic by its employment in general service work. Otherwise, the combined anxiety of the patient and technic may make it impossible for him to secure the desired result. It is entirely natural and laudable to examine and weigh carefully a new method before adopting it to take the place of a technic that is giving routine satisfaction. On the other hand, every patient is entitled to all the available protection against the dangers of aspiration and the pulmonary complications that follow. A technic that removes the field of operation from a supratracheal to an extratracheal position appears to be the only one capable of providing this protection.

REACTION OF ANESTHETISTS

Many anesthetists are shy in adopting intratracheal inhalation anesthesia because of the technic of laryngoscopy. If it were not for the problem of laryngoscopy and of providing the necessary apparatus, intratracheal intubation would be as commonly practiced as a routine use of a pharyngeal tube. Experience alone will overcome this resistance, and adherence to the simple technic suggested will permit the anesthetist to avoid many of the difficulties that the foregoing remarks have suggested. The difficulties of the anesthetist are practically over when intubation has been completed. This experience is so marked and so constant that complicated operations on the nose and throat are now reduced to the simplicity of ordinary abdominal work. The margin of safety is so greatly increased that the anxiety usually attendant in these cases is practically disposed of. My personal reaction to the method may best be summed up by saying that I have never regretted performing intubation on a patient, but that I have frequently regretted a failure to do so.

SUMMARY

Intratracheal inhalation anesthesia built up on the technic of routine laryngoscopy and bronchoscopy rests on the firm foundation of this well established procedure. While trauma is unnecessary in routine bronchoscopy, the possibility of such reaction is reduced to a negligible factor by the relaxation and control of the reflexes secured by the anesthetic. In my hands, trauma has not followed this technic over an experience as a routine method in the last four years. It is believed that this absence of trauma has been partly due to each of the following factors: adequate ether relaxation, elimination of preliminary medication (morphine and atropine), adequate exposure, lubrication of the tube and the use of a mouth gag when intubating. In this technic adequate suction must always be available for the best results. In order to reduce the irritability and the diameter of the intubated element, a pharyngeal gauze pack is preferred to an intratracheal balloon. Irritation is caused by the pharyngeal pack unless care and lubrication are employed in placing it. Experience has shown that the cough spasm following intubation may readily be controlled and its effects promptly met. Technic of intratracheal inhalation provides a functioning equipment for artificial respiration in the case of respiratory failure. Routine cocainization has been avoided, ether relaxation being employed instead. While any anesthetic agent may be employed, ether is the anesthetic of choice in the majority of cases. The opinion appears to be that intubation carried out in the manner prescribed does not predispose to laryngeal tuberculosis in a case with pulmonary tuberculosis, and that the presence of the intratracheal tube through the facilities afforded for suction of the trachea provides one of the most efficient means of reducing postoperative morbidity in thoracic operations. It has been found that the method is indicated in all operations on the head and neck, and in any condition in which aspiration is to be avoided. Routine laryngoscopy immediately following tonsil operations should be practiced. The patients do not complain of the presence of the tube following intubation. The surgeon accepts the technic in all nasal cases, but finds it necessary to cooperate with the anesthetist in tonsillectomy. The chief difficulty from the anesthetist's point of view is the necessity of adequate relaxation and direct laryngoscopy. Once intubation is done, the margin of safety to the patient, to the surgeon and to the anesthetist is greatly increased. The most complicated operations involving the airway then assume the anesthetic simplicity of uncomplicated operations on the extremities.

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PINS AT THE PERIPHERY OF THE LUNG

OBSERVATIONS BASED ON FORTY-TWO CASES

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By pins at the periphery of the lung we mean pins that, as foreign bodies, have invaded relatively minute bronchi. These cases constitute a class distinct from those in which the pin as a foreign body is lodged in the large bronchi, when bronchoscopic removal is quite simple.

In the records of the Bronchoscopic Clinic there are 372 cases of pins of various kinds in the air and food passages. In 113 of these cases the pins were in the bronchi, and in 42 of the 113 the location of the intruder at the time of removal placed it in the peripheral class. The term "at the time of removal" refers to the fact that this kind of foreign body often works downward after the roentgen film is made and even on the bronchoscopic table. Full data on the cases will be found in the records of the clinics published or about to be published.[1]

In the days before the roentgen rays, pins were found in the lung only at autopsy. In the early days of bronchoscopy, pins in the periphery of the lung were considered beyond the limits of bronchoscopy.[1a] Today the perfection of bronchoscopic technic renders peroral removal possible in about 99 per cent of the cases[2] in which the pin is in the middle or either lower lobe, even when the intruder is in the costophrenic angle with the head in contact with the visceral pleura.

ETIOLOGY

The fundamental cause of pins entering the tracheobronchial tree is, as demonstrated by one of us (C. J.),[3] carelessness on the part of

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1. Jackson, Chevalier: (a) Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Company, 1914; (b) Observation on the Pathology of Foreign Bodies in the Air and Food Passages Based on the Analysis of 628 Cases, Mutter Lecture, 1917, Surg., Gynec. & Obst. **28**:201 (March) 1919; (c) Overlooked Foreign Bodies in the Air and Food Passages, Brit. M. J. **2**:686 (Oct. 17) 1925; discussion by Thomas McCrae and others. (d) Jackson, Chevalier, and Jackson, Chevalier Lawrence: Foreign Bodies in the Air and Food Passages, Philadelphia, W. B. Saunders Company, in preparation.

2. Jackson, Chevalier: Are There Cases of Foreign Body in the Lung Impossible of Bronchoscopic Removal? Laryngoscope **31**:528 (July) 1921.

3. Jackson, Chevalier: The Mechanism of Physical Signs, with Especial Reference to Foreign Bodies in the Bronchi, Am. J. M. Sc. **165**:313 (March) 1923.

adults in putting pins in the mouth, and, in children, imitation of the bad example of their elders. The cause of the travel of the pins toward the periphery of the lung is the ratchet-like action of pins.[4] Under the respiratory elongation and shortening of the bronchi, the pins move in the direction of the head, which is practically always downward. The point resists movement upward by sticking into the bronchial wall; the head does not resist movement because it is not sharp; when the head reaches the smallest bronchus that it can enter, increasing frictional resistance against the movement of the head stops its progress toward the periphery usually close to the visceral pleura (fig. 1). Gravity probably plays a small part in the downward progress, as shown by the fact that pins sometimes work toward the lateral periphery.

It is interesting to note that in almost all our cases of pins in the lung the patients, even the babies, were females. In one of the few cases occurring in a man, the patient was using the pin as a toothpick.

PATHOLOGY

The pathology associated with the presence of pins in the lung having been fully presented elsewhere [1b] need not be fully presented here. The features are that pathologic changes are slow in development as in the case of all metallic foreign bodies that are not obstructive. Researches carried out in the Bronchoscopic Clinic many years before this writing showed that the peripheral bronchi are nearly always sterile and that pins probably carry but few bacteria with them into the peripheral bronchi. The defensive powers of the lung are strongly bactericidal so long as ventilation and drainage are maintained. It seems probable, as we have already shown,[5] that the galvanic action of corrosive metals like steel and brass, of which the pins are made, is also bactericidal. The development of tuberculosis in one of our patients (case 6) during the prolonged sojourn of a pin may have been simply a coincidence. It was formerly thought that "phthisis pulmonalis" was a common termination of the prolonged sojourn of a foreign body, but the opinion in those prebacteriologic days was based solely on clinical features.

Pins with heads, such as the common pin or the glass-headed or composition-headed pins, always lodge in the head-downward position in the lung. The area obstructed by the head of a pin is small; hence

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4. Jackson, Chevalier: (a) Prognosis of Foreign Body in the Lung, J. A. M. A. **77**:1178 (Oct. 8) 1921; (b) Bronchoscopy and Esophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927; (c) footnote 1 a.

5. Jackson, Chevalier: (a) Indications, technique et résultats de la bronchoscopie, Monographies Internationales, Paris, Presses Universitaires de France, 1924; (b) footnote 1 a; (c) footnote 1 b.

the pathologic changes are for a long time limited in area. After a time the amount of obstruction increases because of the increase in the size of the pin by corrosion.[6] Eventually abscess develops, and the area of pathologic change spreads wider and wider until the whole lobe or even the whole lung becomes involved (case 6, fig. 8).

The records at our clinic show that common pins invade the posterior branches of the lower lobe stem five times as frequently as the anterior branches. Clinical facts throw no light on the cause for this. Inference from anatomic facts would suggest that recumbency might favor lodgment in posterior branches; but most persons average only one third of the twenty-four hours on the back, and not all of this recumbency is dorsal.

SYMPTOMATOLOGY AND DIAGNOSIS

The initial symptoms, coughing, choking and gagging, are always present, but may be brief and are often overlooked. After the pin reaches a fixed position in the periphery of the lung there are no symptoms for a long time, often six months or more. It is this symptomless interval that misleads the patient and often his physician into the supposition that the patient was mistaken as to a pin having gone down, or into the assumption that the intruder has gone through the gastro-intestinal canal. Sooner or later pathologic changes take place, and the productive cough, hemoptysis and foul expectoration supervene; then follow chilliness, fever, clubbed fingers and toes, watch-crystal nails and all the symptoms of chronic pulmonary sepsis.[7] Symptoms, therefore, in any given case may be absent, and when present, their character will depend on the stage at which the patient is seen.

Roentgenography is the most important diagnostic means, but this does not mean that other methods should be neglected. Roentgenography is invaluable for localization, but accurate interpretation, is not always easy and serious errors have been made (case 1, fig. 1). In some instances it is difficult to determine which lobe is invaded. In all this work stereoscopic roentgenograms are useful, but anteroposterior and lateral films made at 90 degrees from each other are necessary also. The lateral roentgenogram shows the costophrenic angle clearly (figs. 1 and 6), and is essential to prevent the diagnostic error of supposing the pin to be in or below the diaphragm, as occurred in case 1 (fig. 1).

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6. Manges, W. F.: Pathologic Changes in Lung Tissue as the Result of Foreign Bodies of Long Sojourn, J. A. M. A. **87**:987 (Sept. 25) 1926; footnote 1 a, b and d.

7. Jackson, Chevalier: Suppurative Diseases of the Lung Due to Inspirated Foreign Body Contrasted with Those of Other Etiology, Surg., Gynec. & Obst. **42**:305 (March) 1926; footnote 1b and c.

Of the physical signs, a most important early one is limitation of expansion on the invaded side as discovered by Thomas McCrae.[8] Later it is still present but not so valuable or curious, because then pathologic changes sufficient to produce limitation of expansion have supervened. Limitation of expansion is unassociated with bronchial obstruction in those cases in which the pins have been in the bronchi only a short time. All physical signs later are due to the pathologic changes and not to the pin, and are those common in suppurative diseases of the lung.

COMPLICATIONS; SEQUELAE; ASSOCIATION WITH OTHER DISEASES

As these pins are usually close to the pleura, pleuritis is a possible complication, and this may occur either with or without a pneumothorax. When the latter occurs, it may be due to perforation by the foreign body or to the tearing of the pleura by the counter-traction of an old adhesion. If there has been no infection, effusion will be absorbed, and the lung will expand. If, however, the fluid is infected, empyema is inevitable. This will require external drainage, and convalescence will be prolonged. The utmost care should therefore be taken to avoid driving the foreign body through the visceral pleura, as it is certain to carry infection with it.

The sequela in cases of prolonged sojourn is extensive pathologic changes in the entire lung with corresponding ill health and pulmonary suppuration which, if the pin is not removed, ultimately prove fatal. In the cases of bronchoscopic removal there are no pathologic sequelae. The perfect restoration of function in a very much diseased lung is one of the most remarkable demonstrations of the recuperative power of the lung, once its defensive powers are restored by removal of the intruder.

There is no logical reason why a person with pulmonary or other disease might not get a foreign body in the lung. As a matter of clinical fact, however, the association is exceedingly rare. We have had one case of a pin in the periphery of the lung in a tuberculous patient. The tuberculosis was not known to have been, and probably was not, present when the pin was inspirated twenty-four years before. In two other cases tuberculosis was associated, though not etiologically. In the pre-bacteriologic days it was thought that all cases of foreign body in the lung terminated in phthisis pulmonalis.

TREATMENT

Prophylaxis.—The inspiration of a pin into the lung is, in adults, due to the careless and reprehensible habit of putting pins in the mouth

8. McCrae, Thomas: Clinical Features of Foreign Bodies in the Bronchi, Lumeleian Lecture, Lancet **1**:735 (April 12) 1924; 787 (April 19) 1924; 838 (April 26) 1924.

to hold them temporarily. In one case the pin was being used as a toothpick. Children put pins in the mouth in imitation of their elders. For instance, a mother dressing her child holds pins in her mouth; the child when dressing her doll does the same thing as the right and proper way. The prophylaxis in case of both adults and children is obvious. This important subject was extensively considered in previous publications,[9] and all of the conclusions therein reached have been abundantly corroborated by subsequent experience.

A not unimportant point in prophylaxis for the bronchoscopist to remember is to avoid dislodging downward a pin that has temporarily lodged above the larynx. All attempts at removal of pins in this location should be done with the patient on a table steeply inclined toward the head, so that if dislodged the pin will drop into the nasopharynx instead of into the larynx and trachea. Once the latter location is reached, travel toward the periphery by ratchet-like action is independent of gravity and is usually rapid.

Treatment.—No treatment other than peroral bronchoscopic removal is worthy of a moment's consideration. It is rather surprising to note in relatively recent foreign literature that thoracotomy is still being performed for pins and other inspirated foreign bodies at the periphery of the lung. That the difficulties of bronchoscopic removal as compared with foreign bodies in larger bronchi are great is evidenced by the fact that of the last 42 cases of pins at the periphery of the lung unsuccessful bronchoscopies had been done elsewhere before admission in all but 4; but it is equally true that by modern methods, using adequate and modern equipment, in all of these cases the pin was removed at our bronchoscopic clinic without mortality.

Bronchoscopy through the mouth is successful in almost 100 per cent of the cases when pins are located in the middle and lower lobes and descending branches of the upper lobes. The ascending branches of the upper lobes fortunately are rarely invaded at the periphery. Pins in the stem of the upper lobe can be removed even though they are located "around the corner."

We are not now considering pins located in the larger bronchi where the point-sheathing method is easily accomplished under guidance of the eye. At the periphery of the lung the technic is exceedingly difficult but always successful if the problems presented are fully realized, proper care is taken and a proper equipment is available.

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9. Jackson, Chevalier: (a) Foreign Bodies in the Larynx, Trachea and Esophagus Etiologically Considered, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1917, p. 36; (b) What Does Your Baby Put in His Mouth? Hygeia **1**:561 (Dec.) 1923.

It must be realized that the pin in these cases is not free to move; it is almost always fixed, because the ratchet-like action of the pin, under the elongation and shortening of the bronchi during respiration, has buried the point in the wall of the tiny bronchus invaded. Moreover, the head is usually so close to the pleura that the pin cannot be pushed downward. In these cases the costophrenic bronchoscope (fig. 5) will solve the problem. In some cases the standard rotation forceps are used to double the pin into the mouth of the tube; in other cases the pin-bending forceps (fig. 4) may be substituted. The fundamental principle involved is expressed in the axiom of one of us (C. J.) [18] regarding pointed foreign bodies: "Advancing points perforate; retreating points do not." By bending the pin into the costophrenic bronchoscope or the cannula of the pin-bending forceps, the pointed as well as the head end of the pin is caused to retreat (fig. 3). This method must not be used on needles or steel shawl-pins, which would break without bending.

A pin of many years' sojourn in the lung may be so corroded as to break up into many fragments when grasped. The only problem in such a case is the finding of the fragments (case 9).

Fluoroscopic Pathfinding.—In all cases of pins or other foreign bodies located in a bronchus large enough to enter with the bronchoscope, the use of the fluoroscope is an undesirable and, unless carefully used, a dangerous encumbrance in bronchoscopic technic. Of course, with proper care as to screening and dosage, there need be no danger of roentgen-ray burns; the dangers are connected with work in darkness when perfect asepsis and assistance are difficult to maintain, when the patient cannot be perfectly watched and, especially, when control of instruments is based on observation in a fluoroscopic screen, on which great vessels and other vital structures may cast no shadow. The operator may be able to see the foreign body and his instrument, but usually the intervening tissues, however vital they may be, are invisible. When we speak of a fluoroscope for bronchoscopy we mean a double-plane fluoroscope, that is, one that will present on the screen promptly, at will, either the anteroposterior or the lateral projection without rotation of the patient with the bronchoscope in situ, not only because of the possible slight risk of the trauma but because displacement of the relation between the foreign body and the tube-mouth would be inevitable and would defeat all efforts at accurate placement of forceps. A single-plane fluoroscope is altogether unsuitable for bronchoscopic aid because it is misleading. The object may appear to be right at the jaws of the forceps yet actually be as much as 4 or 5 cm. anterior or posterior to it. The use of parallax as an aid in the determination of proximity in the other plane is of comparatively little value. The method of work that has served us well is to place the closed forceps

jaws in apparent contact with the pin in one plane; this procedure is repeated three or four times before the jaws of the forceps are allowed to open. At each change of point of view, from lateral to anteroposterior and vice versa, some displacement is usually found to have occurred. Replacement is not usually to be made by transverse movement; the forceps are slightly withdrawn and then advanced in a direct line with the object; sometimes the line is not direct; it all depends on finding the invaded branch bronchus, so that the forceps will advance without resistance down to the pin. The utmost patience and gentleness in the pathfinding are necessary; any heavy-handed plunging about and tearing out of tissue, as though digging a way down to the foreign body, will meet both defeat and disaster. When, after rechecking many times, the position of the closed forceps is found to be contactual, the jaws are allowed to open and a grasp for the foreign body is made. Usually it will be found that the forceps have not grasped it. It then becomes incumbent on the fluoroscopist to tell the bronchoscopist whether the forceps have closed anteriorly or posteriorly, right or left of the foreign body. The whole maneuver of pathfinding placement is then repeated and again repeated until one is successful in grasping the foreign body. With ordinary bronchoscopes and ordinary forceps it will usually be found that one has grasped the shaft, leaving the point standing out like a barb, preventing withdrawal. With the costophrenic bronchoscope and standard rotation forceps, the pin when grasped by the shaft can be buckled into the tube-mouth or cannula of the forceps, causing *both* pointed and headed extremities to retreat (fig. 3). The difficulties are great, but eventual success follows careful and, if necessary, repeated efforts (W. F. Manges). With the fluoroscopic apparatus devised by Dr. W. Edward Chamberlain for our clinic at Temple University Hospital, about forty-five minutes of work on the fluoroscopic table are within perfectly safe limits of roentgen-ray exposure. The rays are not on continuously, and the exposure is alternately on the side and the back for a few seconds or minutes at a time, and not always at the same spot. We have never yet seen the slightest sign of an erythema. Visibility is a fundamental requirement. Unless the foreign body can clearly be seen in both planes, accurate, safe and successful work is almost impossible. In all of this work the trained eye of the roentgenologist is invaluable, and he should be depended on not only for dosage but for the utmost visibility. In the cases of pin in the left lower lobe, visibility in the anteroposterior diameter can be greatly increased by inflating the stomach with air pumped in with a hand ball attached to a swallowed feeding tube, or it may be pumped in through the esophagoscope. Sometimes, as in the patient shown in figure 2, the air enters the stomach spontaneously.

REPORT OF CASES

Many of our cases have been reported.[10] The following cases are selected as demonstrating additional points of importance. With one exception, in all the cases there had been one or more unsuccessful attempts at bronchoscopic removal prior to admission. Mention of these failures in the respective cases has been omitted; we do not believe that they had anything to do with the progress of the pins toward the periphery; this travel was due to the ratchet-like action already referred to in this and in previous publications.[1a]

CASE 1.—*Laparotomy had previously been done for a pin in the lung, supposed to be in the stomach. Pin removed by peroral costophrenic bronchoscopy. Cure.*

A girl, aged 3 years, came with a history of "swallowing" a common pin while lying on her back, three months prior to admission. The child was taken to a hospital in a distant city where the pin was thought to be in the stomach, on the basis of an anteroposterior roentgenogram, and the absence of pulmonary symptoms. A gastrotomy was done, with negative results. Because of the absence of symptoms after a three months' sojourn, it was supposed that the pin was encysted in the diaphragm. The child was taken to another roentgenologist who made a lateral roentgenogram demonstrating that the pin was in the costophrenic angle of the left lung posterior to the stomach. The patient was then brought to the Temple University Hospital where this localization was verified by Dr. W. Edward Chamberlain (fig. 1). On admission there were no symptoms. The physical signs were negative except for limitation of expansion on the left (invaded) side.

Peroral Costophrenic Bronchoscopy.—The 3 mm. costophrenic bronchoscope was inserted through the mouth and insinuated by careful pathfinding down to the pin. The point was found buried for a distance of about 1 cm. The pin was grasped with rotation forceps near the center, doubled into the tube, as shown in figure 3, and removed through the costophrenic bronchoscope.

Progress.—There was no reaction after bronchoscopy. Roentgen examination gave negative results. The patient returned home in a normal condition two days later.

Comment.—The supposition that the pin was in the stomach and the abdominal section for its removal were based on an anteroposterior roentgenogram alone. The value of the lateral roentgenogram in these costophrenic cases was demonstrated by the second roentgenologist consulted. Both anteroposterior and lateral roentgenograms are always necessary in every case of foreign body, not only for diagnosis and localization but often for the solution of the mechanical problem also. In case of a pin at the periphery of the lung, the lateral view is the only way of determining the vital fact of distance from the pleura. Because

10. Jackson, Chevalier, and Coates, George M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929; Foreign Bodies in the Air and Food Passages, Ann. Otol., Rhin. & Laryng. **33**:1009 (Dec.) 1924; Endoscopie et chirurgie du larynx, Paris, Gaston Doin, 1923; footnote 1 a and c; footnote 4 a and b; footnote 5 a.

of the absence of symptoms, it did not seem possible that the pin could be in the lung, and the inference had been that the pin was encysted in the diaphragm. The explanation is that the symptomless interval was still present at the end of three months. In the pathfinding in this case we had the invaluable aid of Dr. W. Edward Chamberlain, the roentgenologist.

The proximity of the head of the pin to the pleura precluded the possibility of pushing the pin downward to liberate the point from the tissues where it was buried. Any attempt to do so would have sent the pin through into the pleura, with resultant empyema, thoracotomy for drainage, a long tedious convalescence and months of pleural suppuration. The doubling method of causing the point to trail obviated this danger.

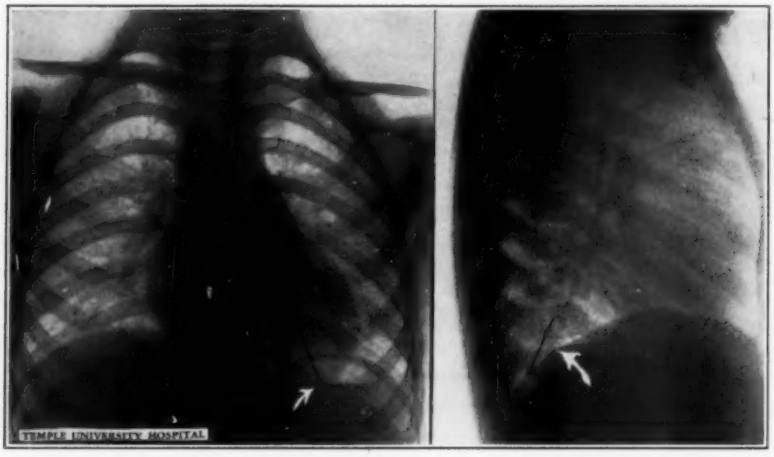

Fig. 1 (case 1).—Roentgenograms, anteroposterior and lateral, of a girl aged 3 years, showing a pin in the costophrenic angle of the right lung. Before admission a gastrotomy had been done, with negative results, on the supposition from an anteroposterior roentgenogram that the foreign body was in the stomach. The pin was removed through the mouth by peroral costophrenic bronchoscopy, using the method illustrated in figure 3.

CASE 2.—*Peroral costophrenic bronchoscopic removal of a pin whose head was in contact with the visceral pleura. Cure.*

A girl, aged 17 years, employed at pinning together accounting slips was holding a pin in her lips when she last control of it; it dropped backward and she inspirated it into her lung. Dr. S. C. McSweeny, when consulted, immediately ordered a roentgen examination, which showed the pin to be in the costophrenic angle of the lung with the head in contact with the pleura, which was "outdented" (fig. 2). There were no physical signs except limitation of expansion on the invaded side.

Peroral Costophrenic Bronchoscopy.—The point of the pin was found embedded in the bronchial wall, requiring extreme delicacy in manipulation to avoid per-

Fig. 2 (case 2).—Roentgenograms of a girl aged 17 years with a pin in the costophrenic angle of the lung, the head "outdenting" the pleura. The pin had been worked downward and its pointed end buried in the bronchial wall by the ratchet-like action created by the pin acting as a pawl during the respiratory and tussive elongation and shortening of the bronchi. On the phase of the mechanical problem of bronchoscopic removal, it is to be noted that the pin cannot be pushed downward to release the point because to do so would drive the head through the visceral pleura, creating a pneumothorax and probably empyema. The best solution of the problem is illustrated in figure 3.

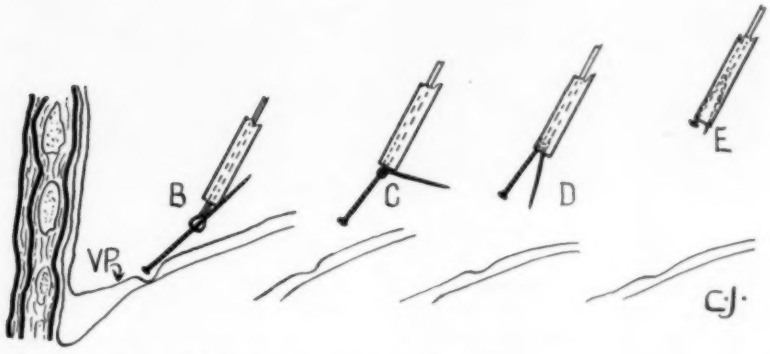

Fig. 3.—Schema illustrating the method of dealing with common pins at the periphery of the lung. By a ratchet-like action the pin has worked itself down until the head indents the visceral pleura (*VP*). The pin cannot be pulled upward because it is anchored by the point buried in tissue; it cannot be pushed downward to release the point, as in the point-sheathing method, because the head would be forced through the pleura. By the method here shown, the head is made to retreat from the pleura and the point is made to retreat from the tissues. The presenting part in these cases is the shaft of the pin. This is seized with rotation forceps (*B*); the pin is then bent into the tiny costophrenic bronchoscope as shown at *C*, *D* and *E*. Sometimes the pin-bending forceps with a larger cannula are used instead of the costophrenic bronchoscope. The mechanical principle is the same. The potentially dangerous advancing point is converted into a trailing point.

foration of the pleura. It was bent into the tube-mouth and removed through the bronchoscope (pathfinding by W. F. Manges).

Progress.—A slight pleuritis followed, but it gave no pain and it cleared without effusion. Complete recovery followed.

Comment.—This case illustrates the tendency of pins to work outward, even to the point where there is "denting" of the visceral pleura. The greatest difficulty is presented by the delicacy of touch required in such cases to allow the pin to move upward during expira-

Fig. 4.—Pin-bending forceps for removing pins from the periphery of the lung. The end of the cannula is of steel, to stand the necessary counterpressure. The proximal portion of the cannula is small so as to permit of respiratory air passing it in the small tubes required for costophrenic bronchoscopy, to supplement the air passing through the glottis alongside the tube.

Fig. 5.—Costophrenic bronchoscope for reaching lesions or pins at the periphery of the lung, especially in the costophrenic angle. The sizes are: 4 mm. by 45 cm. and 2 mm. by 45 cm. for adults; 3 mm. by 35 cm. and 2 mm. by 35 cm. for children, and 2 mm. by 35 cm. for infants. The instrument may be inserted through the laryngoscope, or, in case a larger bronchoscope is used for the proximal part of the tree, it may be inserted through the larger bronchoscope first used, which has been found too large to enter peripheral bronchi in pursuit of a pin that has escaped downward. The rotation forceps smaller than the pin-bending forceps (fig. 4) are used to draw the pin against the distal tube-mouth and thus double it in. The mechanical principle is the same.

tion, if necessary, to prevent perforation by the head. If held too rigidly or if forced downward to the slightest degree, the head would be forced through the visceral pleura.

CASE 3.—*Peroral bronchoscopic removal of a pin from the costophrenic angle of the left lung. Cure.*

A girl, aged 13 years, while on her way to school had two pins in her mouth preparatory to pinning her dress. She threw her head backward in laughing, and inspirated one of the pins. Slight cough and elevation of temperature followed a short symptomless interval.

The physical signs were negative except for limitation of expansion over the lower right portion of the chest.

Roentgen examination revealed the shadow of a pin deep in the costophrenic angle of the left lower lobe, the head almost in contact with the visceral pleura (fig. 6).

Peroral Costophrenic Bronchoscopy.—With the 5 mm. by 45 cm. bronchoscope the pin was removed from a small posterior branch of the left lower lobe bronchus.

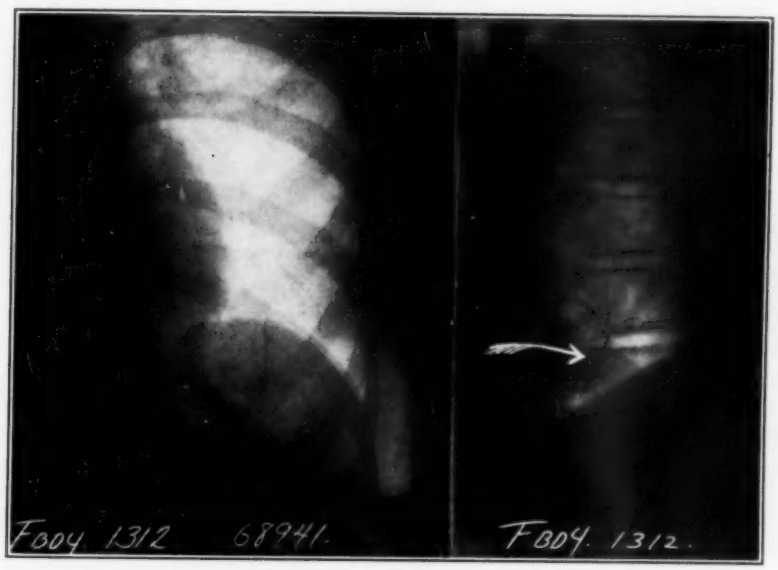

Fig. 6 (case 3).—Roentgenogram of a girl aged 13 years, showing the shadow of a pin in the costophrenic angle of the left lower lobe. The pin was removed by peroral costophrenic bronchoscopy.

Progress.—A slight pleurisy developed, but it cleared up promptly without effusion, and the patient left for home, perfectly well, at the end of a week.

Comment.—The limitation of expansion, greatest over the uninvaded (right) lung and present both before and after the removal of the pin from the other (left) lung, was thought to be due probably to a pleuritis that antedated the inspiration of the pin. This case was exceptional; the usual finding is limitation of expansion over the part of the chest corresponding to the invaded lobe.

CASE 4.—*Pin at periphery of lung for probably thirteen years. Removed by costophrenic bronchoscopy from pathologic tissue, by bending method. Cure.*

A woman, aged 31, complaining of pain, consulted Dr. Grant Bird, who in the course of the studies advised roentgen examination. The films made by Dr. Frederick J. Donovan revealed the shadow of a pin and pathologic changes deep in the lower lobe of the right lung. The patient had no recollection of inspirating the pin, but certain collateral circumstances indicated that the pin had been in the lung for thirteen years.

Roentgen examination at Temple University Hospital confirmed the finding of Dr. Donovan.

Costophrenic Bronchoscopy.—A 4 mm. by 45 cm. bronchoscope was inserted through the mouth into the lower lobe stem until further progress was arrested by the small lumen and the pathologic tissue. The pin-bending forceps (fig. 4) were then insinuated through the passage, and the pin was found to be so firmly embedded in fibrous tissue that it could not be pushed downward to liberate the point; furthermore it was deemed likely that such a movement might perforate the pleura. The pin was then seized as close to the point as possible and bent into the cannula of the forceps. A small portion of the point of the pin broke off, but this afterward came away with pus, blood and secretions.

Progress.—Except for a slight pleurisy of a few days' duration, recovery was uneventful.

Comment.—Sheathing of the point, which is the method of choice in cases of pins not so distally located, was not to be risked here because of the proximity of the pin to the pleura. Doubling of the pin into the costophrenic bronchoscope, accompanied by a correlated slight withdrawing movement, caused *both* points to retreat (fig. 3). It was evident from the details of the history that there had been a long symptomless interval between the initial symptoms and the onset of productive cough, fever and other symptoms, illustrating the clinical fact that sooner or later chronic suppurative disease of the lung supervenes in cases of foreign body, even one so small as a pin.

CASE 5.—*Pin removed from the costophrenic angle of the lung by peroral bronchoscopy. Cure.*

A woman, aged 24, while engaged in pinning accounting slips together habitually held pins in her mouth. One day a pin thus held slipped backward and went down into the tracheobronchial tree, where it soon reached the smallest bronchus that its head could enter in the costophrenic angle of the right lung, as shown by the roentgenogram (fig. 7). The pin was removed by costophrenic bronchoscopy.

Symptoms.—On admission there were no symptoms of any kind, and no physical signs except limitation of expansion over the invaded right lower lobe. There had been choking and gagging and slight cough at the time of the accident, but thereafter the patient was in the symptomless interval.

The diagnosis was made by roentgen examination immediately after the accident and was subsequently confirmed.

Peroral Costophrenic Bronchoscopy.—The smallest bronchoscope was inserted deeply into the lower lobe tree, and the pinpoint was released and brought into the tube-mouth by slightly downward pressure and removed through the bronchoscope, the point-sheathing method.

Progress.—Recovery was uneventful. Perhaps it would be more accurate to say there was nothing from which to recover; the patient was not and had not been ill in any way. The roentgenogram after removal showed no pathologic shadows. The limitation of expansion had practically disappeared.

Comment.—This patient on admission was in the symptomless interval between the complete subsidence of the initial choking, gagging and coughing and the subsequent development of suppuration which gives rise to a productive cough. This interval may be many months; but sooner or later suppuration and ill health develop. The point-sheathing method of removal was barely possible in this case because there was sufficient space to permit of enough downward displacement to release the point.

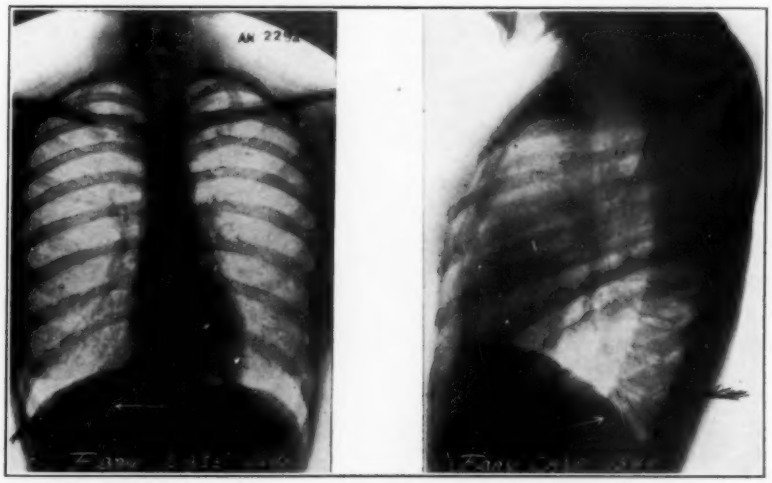

Fig. 7 (case 5).—Roentgenogram of a woman aged 24, showing a pin at the periphery of the lung in the costophrenic angle, behind the liver, 1 cm. from the diaphragmatic pleura. The pin was removed by peroral costophrenic bronchoscopy without general anesthesia and without subsequent reaction. This and many similar cases illustrate the present status of peroral bronchoscopic removal of foreign bodies from the periphery of the lung. In the early days of bronchoscopy foreign bodies in such peripheral locations were beyond the limits of bronchoscopy (Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery).

CASE 6.—*Peroral bronchoscopic removal of a bar-pin of twenty-four years' sojourn in costophrenic angle of lung.*

This case was reported by one of us (C. J.)[18] in 1914 as beyond the limits of bronchoscopy. That it was beyond the limitations of the bronchoscopy of that day was indicated by not merely his own failure but that of two pioneers in bronchoscopy (fig. 8). Twenty-four years later the same patient with the same foreign body came, seriously ill, to Temple University Hospital where, notwithstanding the destructive pathologic changes in the entire lung (fig. 9) due to over twenty years of suppuration, we removed the foreign body in two minutes and

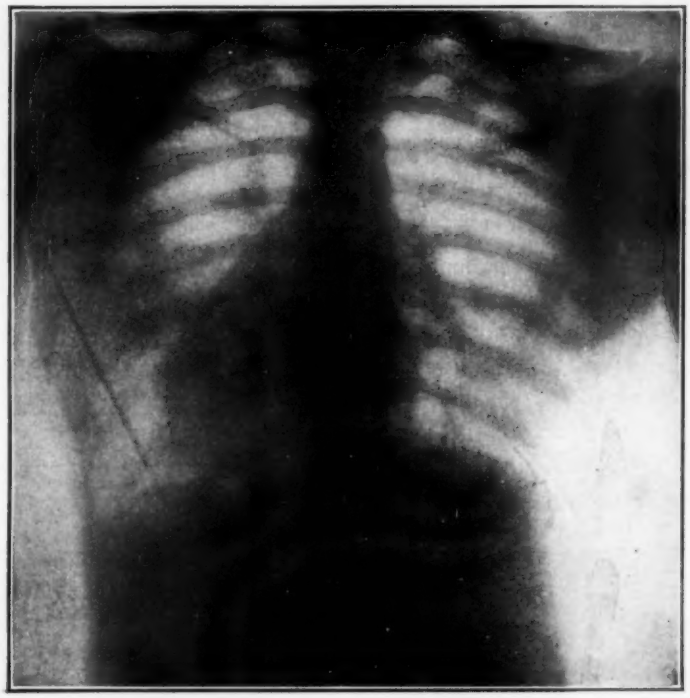

Fig. 8 (case 6).—Roentgenogram of a woman aged 18 years, showing a bar-pin in the extreme periphery (the costophrenic angle of the right lung). This is a reproduction of an illustration published in 1914 (Chevalier Jackson: Peroral Endoscopy and Laryngeal Surgery, fig. 197, lower illustration, p. 322) as depicting a case beyond the limits of bronchoscopy (same patient as in figure 9).

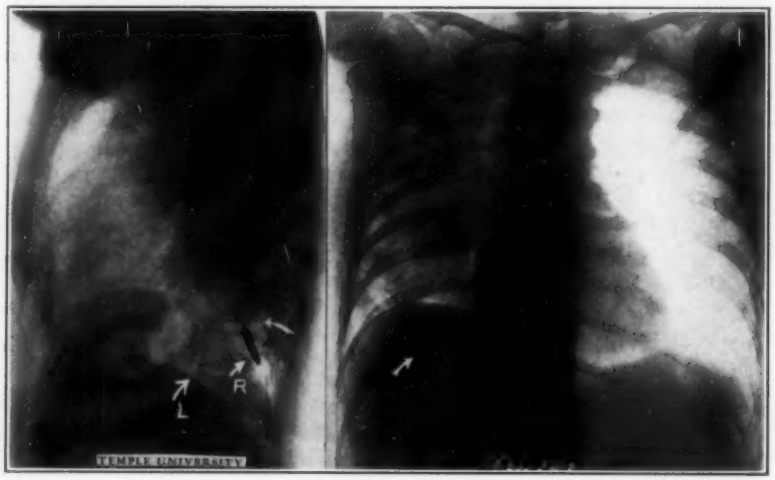

Fig. 9 (case 6).—Roentgenogram of a woman aged 40, showing the shadow of a pin that had been in the lung for twenty-two years, and shadows indicating extensive pathologic changes in the lung. The pin was removed by bronchoscopy in two minutes and twenty-nine seconds. This woman was the same patient with the same pin referred to in figure 8, and reported as beyond the aid of bronchoscopy twenty-two years before. This case is illustrative of the progress of bronchoscopy in the solution of the problem of bronchoscopic removal of pins.

twenty-nine seconds. Nothing could better demonstrate the progress of bronchoscopy in dealing with the problems presented by pins at the periphery of the lung.

CASE 7.—*Removal of a pin by peroral costophrenic bronchoscopy in a baby aged 13 months. Cure.*

A baby girl was being bathed by the mother, who laid several pins on the table. After turning away for a few moments the mother discovered that the child was choking and that thick, blood-flecked saliva was running from the mouth. In a few minutes the baby seemed normal, but the mother consulted the pediatrician, Dr. E. G. Schwartz, who notwithstanding the normal appearance of the child had a roentgen examination made by Dr. X. R. Hyde, revealing the pin (fig. 10). On admission the pulse and respiration were normal, but the temperature ranged to 100 F. Physical examination was difficult and gave negative results except for limitation of expansion low in the right side. No pathologic shadows were visible in the roentgenograms.

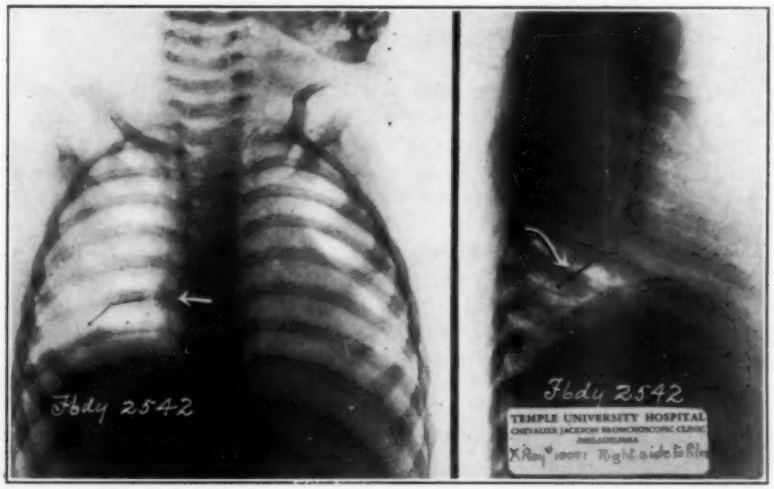

Fig. 10 (case 7).—Roentgenogram of a girl aged 13 months, showing the shadows of a pin that had been in the lower lobe of the right lung for two and a half months. By the time bronchoscopy was done, the pin had reached the costophrenic angle. The pointed half of the pin was buried in tissue up to the point of angulation. The pin was removed by peroral costophrenic bronchoscopy, doubling up the pin so as to make the point trail.

Peroral Costophrenic Bronchoscopy.—When the child was examined fluoroscopically it was found that the foreign body had moved downward and outward from the position shown in figure 10, and had reached the costophrenic angle. The costophrenic bronchoscope and forceps were inserted, the point of the pin was doubled over so as to trail (figs. 3, 4 and 5), and the pin was thus removed harmlessly and without any tendency of the point to catch and become transfixed.

Progress.—There was no reaction from the bronchoscopy. Roentgen examination showed no pathologic shadows. The limitation of expansion had disappeared; the child was discharged well, three days later.

Comment.—By way of prophylaxis it is the duty of every physician to teach mothers the danger of leaving pins within the reach of babies. The bronchoscopic phases of the case are the tendency of pins to work downward and outward toward the periphery by a ratchet-like action, and the advantage of doubling the pin when the pointed end is deeply buried in the tissues near the periphery of the lung. By this method the point is made to retreat from the tissues.

CASE 8.—*Peroral bronchoscopic removal of pin from depths of right lower lobe. Cure.*

A girl, aged 17 years, while looking upward, inspirated a pin held in the mouth during removal of decorations from a wall. After the initial choking and coughing there were no symptoms.

Physical examination revealed limitation of expansion over the entire right side of the chest, especially in the lower portion. The difference in percussion on either side could be made out, and there was no alteration in the signs on auscultation.

Roentgen examination before admission had located the pin in an anterior branch of the right lower lobe bronchus, near the level of the dome of the diaphragm. At our examination it was found somewhat lower.

Peroral bronchoscopy under local anesthesia, with pathfinding assistance by Dr. Manges, resulted in the successful removal of the pin in thirteen minutes and thirty-five seconds.

There was no reaction after bronchoscopy. The temperature remained normal. The pulse rate, which was taken immediately, was increased a trifle for a few minutes. There was nothing from which to recover. Roentgen examination showed no pathologic shadows. The expansion had become equal on both sides. The patient went home the second day after bronchoscopy.

Comment.—This case is a good illustration of the symptomless interval that occurs in all cases of pins in the lung. It also illustrates the peculiar limitation of expansion on the invaded side in cases of nonobstructive foreign bodies in the lung, first noted by Thomas McCrae.[8] Of course, the limitation of motion is noted also in cases of large obstructive foreign bodies, but in them there is nothing remarkable about it. The success of peroral bronchoscopic removal in peripheral cases is again demonstrated.

CASE 9.—*Pin in periphery of left lung for many years. Removal by peripheral bronchoscopy; problem of picking up fragments of disintegrated pin. Cure.*

A woman, aged 40, had a slight but persistent and sometimes paroxysmal cough for many years. Expectoration was purulent but small in amount and occurred only at long intervals. Disease of the nasal sinuses was thought to be the cause, but great improvement of this condition under treatment did not help the cough, which was called "habitual." An internist ordered a roentgenogram, which revealed a pin at the level of the dome of the diaphragm, slanting forward, indicating a location in an anterior branch of the lower lobe stem. The pin was irregular in outline, indicating corrosion from prolonged sojourn. There was no recollection by this patient or her parents of swallowing or inspirating a pin; it was in the search for the cause of a persistent cough that the pin was discovered.

Peripheral Bronchoscopy.—It required a 4 mm. bronchoscope to reach the region of the pin. At this point there was a small purulent fistula in which the pin was located. When the pin was grasped near its point it crumbled into a number of fragments which required separate picking up.

Comment.—This case illustrates the wisdom of the internist in running down every possible cause of cough, rather than accepting the diagnosis of "habitual cough." It also shows that though there may be a long symptomless interval, sooner or later a pin in the lung will cause trouble. The long duration had totally changed the mechanical problem. Instead of having a dangerous point with which to deal, there was the exceedingly difficult problem of picking up many minute fragments of a foreign body in a tiny fistula at the periphery of the lung. It is important to recognize this class of case as totally different from that of the recently inspirated pin that requires bending to make the point trail.

Progress.—There was no reaction. A small amount (1 or 2 cc.) of fetid pus was expectorated once daily, usually in the morning. This gradually ceased. At the end of six months a letter was received from the patient stating, "For the first time in many years I am free from cough that I feared had become a habit."

CASE 10.—*Peroral bronchoscopic removal of a pin from periphery of right lower lobe. Cure.*

A girl, aged 12 years, took a deep breath, forgetting about a pin that she was holding in her mouth while arranging a ribbon which she intended to pin to her dress. She had a slight unproductive cough; otherwise there were no symptoms after the initial coughing and choking. Physical examination gave negative results except for limitation of expansion on the right side. Roentgen examination showed a pin low down in the right lung.

Peroral Costophrenic Bronchoscopy.—At bronchoscopy the pin was found to have moved lower into the extreme depth of the costophrenic angle of the right lung. With a 5 mm. bronchoscope the orifice of the invaded bronchus was reached, and the point of the pin was freed and brought into the tube-mouth and thus safely withdrawn.

Progress.—There was no reaction. Roentgen examination after removal was negative for pathologic changes. The patient returned to her home in an adjoining state on the third day in a perfectly normal condition. The limitation of expansion on the one side had disappeared at the time the patient left the hospital.

Comment.—This is another case demonstrating the value of bronchoscopes of very small diameter in reaching foreign bodies in the periphery of the lung. Again is demonstrated the phenomenon of limitation of expansion on the invaded side in the case of a small, nonobstructive foreign body.

CASE 11.—*Peroral costophrenic bronchoscopic removal of a pin from a child aged 6 years. Cure.*

While playing with a pin, the boy placed it in his mouth and inspirated it. The child was alone at the time of the accident. No symptoms were noted then nor on admission. Physical examination showed lagging of inspiratory movement on

inspection, and quieter breath sounds on auscultation, over the left lower lobe. Roentgen examination showed a pin deep in the left lower lobe.

Peroral Peripheral Bronchoscopy.—It was found that the pin had moved deeper into the lung in an anterior branch of the left lower lobe stem. The orifice of this tiny bronchial branch could be presented, but it was too small (less than 2 mm. in diameter) to be entered with the tube-mouth. The forward grasping forceps were inserted, and by rotation of the forceps the pin was moved downward and freed. With the jaw of the forceps covering the point, safe removal through the bronchoscope quickly followed.

Progress.—There was no reaction; roentgen examination showed no pathologic shadows. The physical signs were normal, the limitation of expansion on the invaded side having disappeared two days after removal of the pin. The child was discharged well.

Comment.—The location of the pin in the anterior bronchus of the lower lobe stem is relatively rare as compared with that in posterior branches (5:1). The disappearance of the limitation of expansion after removal of the pin is the rule in cases in which the pin has been in the lung but a few days; the time required is usually in proportion to the length of sojourn of the pin. Doubling up of the pin was not necessary in this case because there was a sufficient distance between the head of the pin and the pleura to permit of pushing the head downward to free the point.

CASE 12.—*Limitation of expansion owing to presence of pin in lung. Disappearance of limitation after removal of pin by peroral costophrenic bronchoscopy. Cure.*

A woman, aged 21, while dressing in a gymnasium, placed a number of pins in her mouth. While she was laughing, one went down. Roentgen examination showed the pin to be in the left bronchus, where it soon reached the smallest bronchial branch that it could enter; the head was almost in contact with the diaphragmatic pleura. Physical examination gave entirely negative results except for limitation of expansion on the left side. There was an occasional slight cough; it probably would not have been noticed if the presence of the pin in the lung had been unknown.

Recovery was uneventful; in fact there was nothing from which to recover. The limitation of expansion had practically disappeared at the end of the second day.

Comment.—This case is a good demonstration of limitation of expansion in the invaded side only, owing to the presence of a pin. It is not always possible to turn inward and sheath the point of a pin without either pushing it downward or doubling it.

CASE 13.—*Peroral costophrenic bronchoscopic removal of pin from depths of right lung. Cure.*

About two and a half months before admission, the patient, a woman, aged 26, inspirated a pin that she was holding in her mouth. She did not remember any initial symptoms. Recently there had been cough, slightly foul expectoration, dyspnea on exertion and slight wheezing heard at the mouth. Physical examination showed slight impairment of percussion and marked limitation of expansion on the

right side. Roentgenograms showed the pin to be in one of the terminal costophrenic bronchi on the right side (fig. 11).

Peroral Costophrenic Bronchoscopy.—The shaft of the pin presented itself to the forceps because the point was deeply buried in the bronchial wall. The shaft was seized, and the pointed end was bent over against the tube-mouth to cause the point to trail. Removal then was easy and safe.

Comment.—There was some suppuration in the right lower lobe bronchi which accounted for the symptoms and for the signs other than the limitation of expansion, but it is not certain that the suppura-

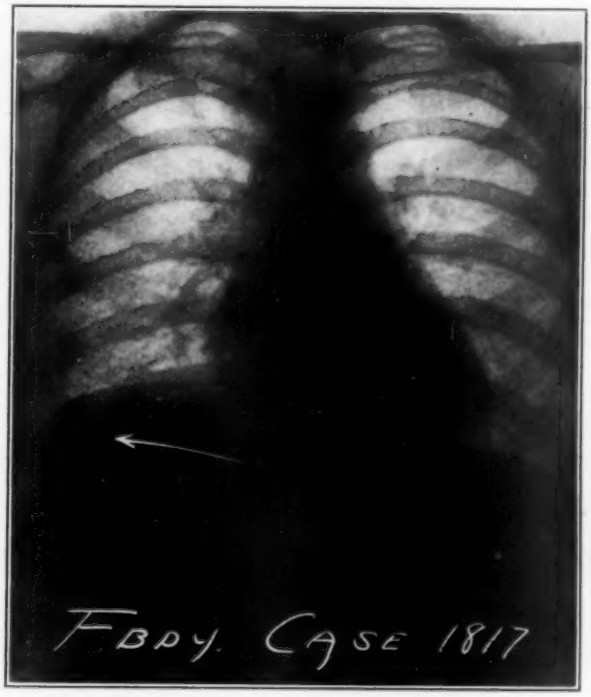

Fig. 11 (case 13).—Roentgenogram of a woman aged 26, showing the shadow of a pin in the costophrenic angle of the lower lobe of the right lung, the entire pin being below the level of the dome of the diaphragm. Removal was effected by peroral costophrenic bronchoscopy, with cure.

tion was a direct result of the foreign body. The longer time required for the disappearance of the limitation of expansion was, as usual, proportionate to the length of sojourn.

CASE 14.—*Peroral costophrenic bronchoscopic removal of pin after two years' sojourn in Scottish boy aged 5 years. Cure.*

The boy in play had been using the pin to imitate the smoking of a cigarette; he lost control of the pin, and it slipped down his throat. A severe attack of coughing immediately followed, but soon subsided; afterward there were no symp-

toms for a number of months. Later, attacks of productive cough developed. Recently there had been slight fever; on admission it was 99 F. Physical signs were slight impairment of percussion, distant breath sounds over the left lower lobe posteriorly and definite limitation of expansion over the left side of the chest.

Roentgen examination showed a shadow of the pin low down in the left lower lobe (fig. 12).

Costophrenic Bronchoscopy.—It was found that the pin had worked deeper and the point had not yet become deeply buried, which rendered it possible to liberate the point with rotation forceps and to sheath it in the tube-mouth and remove the pin. The boy was discharged two days later in good condition; the limitation of expansion was less, but had not altogether disappeared.

Comment.—The relative rarity of foreign bodies in the lung of children in Scotland is curious in view of our records, which show a number of Scottish names about proportionate to the percentage

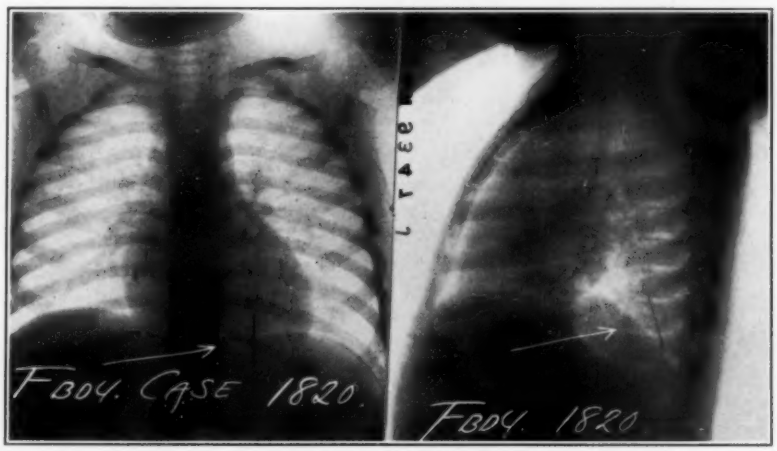

Fig. 12 (case 14).—Roentgenogram of a boy aged 5 years, showing a pin in the costophrenic angle of the lower lobe of the left lung, the head in contact with the visceral pleura. The pin was removed by peroral costophrenic bronchoscopy.

of Scottish people in the population. Possibly, as suggested by A. Logan Turner, the more careful supervision of children in Scotland is a factor. The diminution in the degree of limitation of expansion in two days was remarkable in a case of foreign body of two years' sojourn; unfortunately, we were unable to get accurate data on the time of its ultimate disappearance, though the child was known to be symptom-free at the end of three months.

CASE 15.—*Peroral costophrenic bronchoscopic removal of pin from the right lung of a boy aged 7 years. Cure.*

The child was holding a pin in his mouth in play, when he was struck on the head by a companion. The pin went down his throat, causing gagging and choking. Afterward there were no symptoms except slight cough. On admission to

the hospital, five days later, the physical signs were negative except for limitation of expansion on the right side. Roentgen examination located the pin in the right lower lobe, its axis being in the line of the lower lobe stem (fig. 13).

Peroral Costophrenic Bronchoscopy.—The pin was found to have worked downward into the terminal bronchial branches of the right lower lobe stem in the costophrenic angle. The point was brought into the mouth of the bronchoscope and removed. The child was discharged well the following day. No record was made as to the limitation of expansion after removal of the pin.

Comment.—The spontaneous working downward of the pin in this case is noteworthy because it is one of the very few cases of pins in the periphery of the lung in which there had been no bronchoscopic attempts at removal prior to admission.

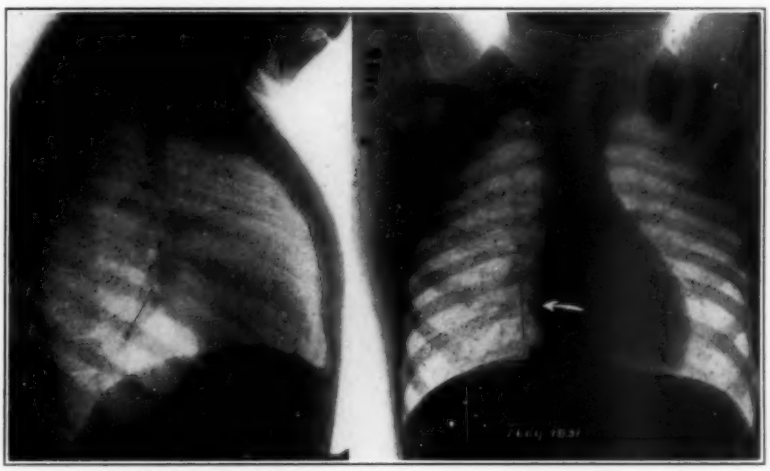

Fig. 13 (case 15).—Roentgenogram of a boy aged 7 years, showing a pin in the stem of the right lower lobe, heading for the costophrenic angle. This angle had been reached by the time the bronchoscope arrived at the pin. Removal by peroral costophrenic bronchoscopy resulted in a cure.

CONCLUSIONS

1. Pins at the periphery of the lung are practically always lodged head downward. This may be due to the catching of the point and the tumbling over of the head end, and partly to the fact that the head end is the heavier.

2. The primary cause of pins in the lung is carelessness in putting pins in the mouth. The numerous secondary etiologic factors are the same as those given in the previously published analysis.[9a] The cause of the pin reaching the periphery is a pawl and ratchet-like action of the pin; the head is free to move downward during inspiratory elongation of the bronchi, and the point catches and resists upward movement during

the bronchial shortening of expiration and cough. The limit of downward travel is the smallest bronchus that the head of the pin can enter.

3. After the initial choking, gagging and coughing there is, in the case of pins at the periphery of the lung, a symptomless interval of a number of months. Sooner or later, however, suppurative changes, with productive cough and progressively increasing impairment of health, supervene, and a fatal ending may eventually occur if the pin is not removed.

4. All pins at the periphery of the middle and lower lobes and descending branches of the upper lobes can be removed through the mouth by peroral costophrenic bronchoscopy. The ascending branches of the upper lobes present great difficulties, but fortunately their invasion by pins is exceedingly rare.

CALCIGEROUS METAPLASIA IN THE AURICLE

REPORT OF TWO CASES

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SANTA BARBARA, CALIF.

Bony deposits in the outer ear are apparently rare, for only thirty-eight cases have been recorded.[1] Although little is known of the etiology of this condition, it is believed that senile changes and injury from frostbite may be of significance. A preceding infection has been recorded in only one case.[2]

REPORT OF CASES

In the following two cases all three of these factors were present:

CASE 1.—A man, aged 71, came to the Sansum Clinic on Jan. 18, 1932, for a check up. General reexamination revealed some cardiac hypertrophy and an early

Figure 1

Figure 2

Fig. 1.—Roentgenogram of the left and right ears, showing deposits of bone in the outer portion of each.

Fig. 2.—Roentgenogram of the left ear, showing one large and two smaller deposits of bone.

decompensation. In the course of the examination of the ear, nose and throat, the helix of each ear was found to be of bony hardness. This was more marked on the right (fig. 1). There was no external deformity of the ear. A keratosis 4 mm. in diameter was present in the scaphoid fossa of the right ear. Aural examination otherwise gave negative results. The tonsils were large and apparently infected. The patient stated that the ears had been frostbitten about a dozen times

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Submitted for publication, March 10, 1932.
From the Sansum Clinic.

1. Higbee, D. R.: Calcigerous Metaplasia of the Auricular Cartilage, Arch. Otolaryng. **14**:70 (July) 1931.

2. Quoted by Higbee (footnote 1).

in his youth. Otherwise, the history was irrelevant. The bony deposits never caused discomfort; in fact, he was not conscious of them.

CASE 2.—A man, aged 72, came to the Sansum Clinic on Nov. 30, 1931, for relief from frequent colds. In his youth the ears had been frostbitten on several occasions. Thirty-five years before examination an attack of erysipelas had involved the whole of the left ear and part of the left side of the face. Twenty years prior to examination a soft, cystic, pea-sized mass appeared rather suddenly in the scaphoid fossa of the left pinna. The swelling was circumscribed and painless. After several months it gradually began to harden, and over a period of a few years it became bony and slowly increased in size. A few years previous to examination another smaller, bony deposit had appeared in the upper portion of the ear adjacent to the principal mass. The patient did not recall having mechanically traumatized the ear. Discomfort rarely occurred, and then only when he lay on that side. The hearing in both ears had been impaired for about twenty years.

General physical examination disclosed an early hypertension on an arteriosclerotic basis. On the skin of the face and neck were a number of small scattered keratoses. On testing the hearing, a moderately advanced combined type of deafness was found. The right auricle was of a normal appearance, but the upper portion of the left ear was moderately thickened by a hard bony swelling situated in the scaphoid fossa and involving the helix. This was nodular and irregularly oval, the vertical diameter being the longest. In the upper portion of the helix was another smaller area of bone. The cartilage of the lower portion of the antihelix, for an area 1.5 by .4 cm., though not bony, was quite firm. Two small keratoses were present on the skin of the helix, and there was another on the skin over the anterior surface of the larger bony mass. The skin was not ulcerated or adherent. Roentgenograms of the ear were made (fig. 2). No treatment for the condition seemed to be indicated.

COMMENT

It is probable that bony deposits in the auricle are of more frequent occurrence than is generally believed. Fraenkel [2] found, on roentgenologic examination of seventy-five persons, that there was calcification or ossification in the cartilage of the auricle in fifteen. The formation of bony deposits seems to depend on senile changes and frostbite. The preceding infectious process that occurred in case 2 probably had some influence on the formation of the bony mass, as it was quite nodular and thickened, unlike the flat bone present in case 1.

SUPPURATIVE MENINGITIS OF OTITIC AND NASAL ORIGIN

ITS RELATION TO BLOOD STREAM INVASION OF THE PIAL VESSELS

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During the past few years, 367 persons with suppurative meningitis originating from the ear or nose were admitted at the Newark Eye and Ear Infirmary and the Newark City Hospital. Two hundred and thirteen patients were operated on; of these, 145 died, 68 recovered (about 32 per cent), and 105 autopsies were performed.[1]

This report is an attempt to summarize this experience and to add what knowledge has been acquired since my last reports.[2] Its chief value lies in the surgical information obtained from the autopsies, each autopsy having been conducted to ascertain (*a*) the particular lesion present, (*b*) what had been accomplished by the treatment and (*c*) whether further or different surgical measures might not have resulted in the patient's recovery.

SUBARACHNOID SPACE MENINGITIS VERSUS MENINGITIS SECONDARY TO PHLEBITIS OF ARACHNOID VESSELS

Surgically, there are two types of meningitis, depending on (*a*) the mode of entrance into the meninges and (*b*) the pathway by which the infection extends.

Subarachnoid Space Meningitis, Which Originates by Direct Extension from a Primary Focus of Suppuration in the Ear or Nose.—The bacterial infection is confined to the subarachnoid spaces, at first to that part adjacent to the primary focus. The inflammation advances

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Presented in part before the Section on Laryngology, Otology and Rhinology, at the Eighty-Second Annual Session of the American Medical Association, Philadelphia, June 11, 1931.

1. The large number of cases has resulted from the coordination of the cranial surgery department with the otologic department. The autopsies and pathologic examinations were by Drs. Lewis Brown, Harrison S. Martland and Asher Yaguda and Mr. David McKinnon.

2. Eagleton, Wells P.: (*a*) Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926; (*b*) Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, Arch. Otolaryng. **13**: 386 (March) 1931.

within the arachnoid mesh. In the terminal stages of subarachnoid space meningitis, the blood vessels of the arachnoid may be attacked.

Subarachnoid space meningitis is curable by surgical methods. Success depends on drainage of the infected areas; it must be instituted while the infected fluid is localized in a cistern adjacent to the primary focus in the bone (localized subarachnoid space meningitis) or within a prolongation connected with an end-organ, such as the membranous labyrinth [3] (localized subarachnoid space meningitis within the prolongation of the internal auditory meatus). However, even after the septic process has become diffuse (general leptomeningitis), a small proportion of patients recover, provided the blood vessels within the arachnoid are not attacked.

Arachnoid Vessel Meningitis, Namely, Meningitis Secondary to a Septic Infection of the Blood Vessels of the Arachnoid.—In this type the inflammation enters the meninges by the blood vessels and is carried by them into different parts of the arachnoid. Success in the treatment of meningitis from infection of the arachnoid vessels has not been attained, at least not after the process is well advanced. However, the early removal of the primary focus of suppuration (including the eradication of all infected vessels) while the phlebitic process in the meningeal vessels is confined to an area adjacent to the suppuration in the temporal bone, is followed by the immediate disappearance of the profound cerebral symptoms that invariably accompany invasion of the soft meninx by infected blood vessels.

OPERATIVE TECHNIC IN PNEUMOCOCCIC MASTOIDITIS WITH BEGINNING MENINGEAL SYMPTOMS

All the bone of the mastoid and squama which covers the dura and is not a part of the labyrinth capsule is sacrificed, thus removing any area of osteothrombophlebitis that may be infecting the meninges. At the same time, the lateral cistern in the neighborhood of the saccus endolymphaticus is opened, as is also the saccus if infected.[2b]

The results have been most gratifying, even when isolated pneumococci have been found in the lumbar fluid. In cases in which meningeal symptoms were accompanied by cloudy but sterile fluid from the lumbar region, the operation was followed by an immediate disappearance of all alarming symptoms. In numerous instances the fluid evacuated from the arachnoid cistern contained pneumococci, although the fluid from the lumbar puncture was sterile.

TENTATIVE DIAGNOSIS

Shortly after admission, each patient with suppurative meningitis is operated on, excepting when in extremis, or when he has the blue

3. Eagleton, Wells P.: The Operative Treatment of Suppurative Meningitis: A Second Report of Clinical Experience in Subarachnoid Irrigation, and of Postmortem Observations, Atlantic M. J. **26**:575 (June) 1923.

color arising from a profound cerebral intoxication (which I regard as significant of multiple punctate hemorrhage in the cortex). Before operating, however, a tentative diagnosis is attempted based on the determination of (*a*) the time that the infection has existed within the dura, (*b*) the route by which it entered and the probable location of the septic fluid, that is, what specific arachnoid cistern is infected. Recently, I have come to recognize that from a prognostic standpoint, it is of the highest importance to diagnose (*c*) the type of meningitis, whether (1) it is subarachnoid space meningitis or (2) meningitis secondary to infection of the arachnoid vessel.

The tentative diagnosis is based on facts elicited by:

1. A chronological history of the date of the appearance and the duration of all symptoms. The symptomatic inquiry is continued day by day from the inception of the aural or nasal suppuration to the time of the examination.

2. A general neurologic examination, which includes testing of (*a*) the cochlear apparatus and of (*b*) the vestibular mechanisms, (*c*) an ophthalmoscopic examination of the fundi and (*d*) a perimetric examination of the visual fields.

3. Laboratory findings in (1) an examination of the cerebrospinal fluid, consisting of an estimate of (*a*) the pressure, (*b*) a cell count and (*c*) a careful search for bacteria; (2) a differential and bacterial examination of the blood, and (3) a bacterial examination of the aural discharge.

4. Roentgen studies (*a*) of the mastoid and (*b*) of the petrous apex in aural cases, and (*c*) of the accessory sinuses in nasal suppuration.

INTERPRETATION OF CEREBRAL SYMPTOMS IN MENINGITIS

In a general way, in the presence of an aural or nasal suppurative lesion, (*a*) any headache combined with signs of cerebral involvement is suggestive of meningitis. (*b*) Vomiting is regarded as the expression of a cerebral reaction. In the construction of a mental picture of the chronological course of the disease, the first vomiting is regarded as clinical evidence of the date when the infection entered the meninges. (*c*) A convulsion in an adult or a "sinking spell" in a child is suggestive of a sudden vascular insult to the brain, usually an infarct with occlusion of a cortical vessel. They occur when the cerebral blood stream has been suddenly attacked. Both are of grave prognostic significance. (*d*) A rigor is evidence of general blood stream infection. (*e*) Drowsiness, when associated with a slow pulse and papilledema, suggests that the meningitis is secondary to a brain abscess. (*f*) Continued drowsiness, with a normal fundus or with slight blurring of the

disk, in the presence of a blood stream infection and a rapid pulse rate, is suggestive of cerebral edema from encephalitis.

Cerebrospinal Fluid.—(*a*) Markedly increased pressure of the fluid, with a normal cell content, is interpreted as the expression of a protective reaction. The infected bony area causing the irritation must be adjacent to, or in communication with, the arachnoid mesh. (*b*) A cell count of over 100 is suggestive of a beginning localized meningitis, provided, of course, there is a suppurative process in the ear or nose and there are other signs of meningitis—fever, headache, etc. (*c*) A moderate number of micro-organisms in the fluid is evidence of a generalized septic leptomeningitis. It is of alarming import, although a cloudy fluid containing a few organisms may occur as an overflow process, if the chief collection of infected fluid is still limited to the cistern adjacent to the primary focus in the bone. In the latter case the patient gives a "favorable impression."

(*d*) Clear fluid containing numerous organisms is of the gravest prognostic significance as it occurs in cases of rapidly extending suppurative meningitis. (*e*) Yellow fluid (xanthochromia) is diagnostic of old hemorrhage into the cerebrospinal system. It occurs in spontaneous noninfective subarachnoid hemorrhage into the cistern of the base. It has been mistaken for an infected fluid, especially as subarachnoid hemorrhage of the base is associated with a moderately high temperature and a stiff neck.

Interpretation of the Blood Examination.—(*a*) A positive blood culture is evidence of a blood stream infection. (*b*) A positive culture associated with a steeplechase temperature, especially in the presence of repeated chills, is evidence that the infection is still confined to a venous sinus, even when the cerebrospinal fluid pressure is elevated and there is a moderately increased cell count. In this case the meningeal reaction is regarded as protective. (*c*) Early and repeated positive blood cultures associated with a continuously high temperature and meningeal symptoms are suggestive of (1) thrombophlebitis of the cerebral veins, or of (2) an acute hematogenous osteomyelitis of a cartilaginous bone (the sphenoid or petrous apex) adjacent to the meninges.

LOCALIZING SYMPTOMS IN SUPPURATIVE AURAL CASES

In a general way, (*a*) a stiff neck signifies involvement of the posterior fossa.

(*b*) A nonreacting labyrinth also suggests the posterior fossa as the seat of the disease, as does (*c*) a spontaneous nystagmus of cerebral origin.

(*d*) Vertical nystagmus in meningitis is a terminal manifestation.

(*e*) Pain in the distribution of a branch of the fifth nerve or lessened sensibility of the homolateral cornea shows invasion of the middle fossa. In my experience, the corneal neuranesthesia like the neuralgia may be transient.

(*f*) The "significant pain behind the eye" associated with a low grade sepsis, in the presence of recurrent attacks of stiff neck and pain in the head, with or without abductor paralysis, is diagnostic of bulbar cisternal meningitis, the result of caries of the petrous apex.[4]

(*g*) Hemianoptic indentations of the visual fields furnish evidence of a collection of fluid in the chiasmal cistern from suppuration of the petrous apex.[5]

(*h*) Blindness in the absence of ophthalmoscopic change suggests pus in the ventricle.[6]

(*i*) Hypersensitiveness of the auricle with meningitis suggests that there is a deep bony lesion within the middle fossa. I have seen it occur in osteomyelitis of the petrous apex.[7]

(*j*) Sudden contralateral motor paralysis associated with evidence (1) of venous infection (chills) and signs (2) of profound toxemia (high temperature and great prostration) suggests thrombophlebitis of the great sylvian vein.

Induced Vestibular Reaction.—(*a*) Absence of reaction from caloric stimulation of the ear opposite to the suppurating one when the patient is in the upright position, if followed by a prompt horizontal nystagmus when he assumes the recumbent position, signifies increased intracranial pressure.[8] (*b*) Reduction of the duration of the induced nystagmus,

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4. Eagleton, Wells P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386 (March) 1930.

5. Eagleton, Wells P.: Sixth Nerve Involvement in the Diagnosis and Surgical Treatment of Meningitis of Otitic and Nasal Origin, Tr. I., Congrès international d'oto-rhino-laryngologie, Copenhagen, 1928, p. 822.

6. Malis, S.: Pneumobacillus (Friedländer's) Meningitis Secondary to Otitis Media (Case 2), Arch. Otolaryng. **12**:162 (Aug.) 1930.

7. Eagleton, Wells P.: Case of Recrudescence of a Latent Infection (Streptococci) in the Marrow Substance of the Petrous Apex and the Sphenoidal Sinus: Immediate Blood Stream Infection, Diffuse Thrombophlebitis of Cerebral Veins; Basilar Meningitis; Cerebral Compression from Encephalitis, Edema of Brain, and Nonobstructive Internal Hydrocephalis, to be published.

8. Eagleton, Wells P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin: Report of Three Cases with a Previously Undescribed Aural Symptom of Increased Intracranial Pressures, Tr. Am. Otol. Soc. **12**:564, 1912.

which normally follows rotation by at least 50 per cent, suggests effusion into the pathways of the cerebrospinal system over the cerebellar cortex.[9]

(*c*) Nonreactibility to caloric stimulation of both ears with the patient in the vertical and horizontal positions, when combined with failure of all turning reactions, although he hears in one or both ears, occurs in effusion into the arachnoid mesh covering the anterior surface of the pons and medulla and the inferior surface of the cerebellum (bulbar cisternal meningitis). (See illustration.)

Localized bulbar cisternal meningitis in which the exudate was limited to the arachnoid mesh over the brain stem and pons and within the inferior collar of the cerebellum. There was absence of all caloric and turning reactions, with hearing in one ear. The general symptoms in this patient were a rise in temperature, stiff neck, falling and a Kernig sign, but no headache, dizziness or nystagmus.

LOCALIZING SIGNS OF MENINGITIS FROM FRONTAL, ETHMOIDAL AND SPHENOIDAL SINUS SUPPURATION

Frontal and Ethmoidal Sinus Suppuration.—(*a*) Euphoria is suggestive of an accumulation of irritating fluid over the frontal lobe. (*b*) Third nerve paralysis in ethmoid disease suggests invasion of the chiasmal cistern.

9. Eagleton, Wells P.: Importance of the Absence of the Reactibility of the Vertical Canals, in Increased Intracranial Pressure and the Reduction of the Duration of the Nystagmus from Turning in Protective Meningitis, Tr. Am. Laryng., Rhin. & Otol. Soc., 1922, p. 231.

Sphenoidal Sinus Suppuration.—(*a*) A history of previous attacks of pain at the top of the head suggests a chronic sphenoidal empyema.

(*b*) In my experience, acute pneumococcic type III meningitis following a cold in the head is suggestive that the suppuration in the sphenoidal sinus may be the primary focus of infection (unless the meningitis is a complication of pneumonia or has followed a pneumococcic otitis with mastoiditis, which has extended by way of the eustachian tube). (See embolic otitis.)

LOCALIZING SIGNS IN A COMBINATION OF AURAL AND SPHENOIDAL SUPPURATION

(*a*) Suppurative meningitis preceded by a long period of ill health, and when associated with early and repeated positive blood cultures, without a thrombus in a dural sinus, suggests osteomyelitis of the sphenoidal base as the cause of the meningitis.

(*b*) The immediate presence of a positive blood culture in pneumococcic meningitis without pneumonia suggests the sphenoidal sinus as the primary focus, even in cases of aural suppuration, when investigation of the neighboring venous sinuses—the lateral and petrosal—and of the jugular bulb fails to reveal thrombophlebitis.

SUBARACHNOID SPACE MENINGITIS

Meningitis that originates by direct extension from a primary focus of suppuration in the ear or the nose at first is localized within the cerebrospinal fluid system. The site of the primary focus in the bone or end-organ or in a venous sinus dictates the particular cistern or the part of the arachnoid mesh that is first invaded.[10]

Basal, Cortical and Ventricular Subarachnoid Meningitis.—When the suppuration is confined to the basal cisterna, there is (*a*) localized or general basal meningitis; when the fine arachnoid channels covering the cortex are attacked, there results (*b*) cortical or "Hut" meningitis. Finally, the infection may invade the ventricular system—the choroid plexus and the ependyma—with a resulting (*c*) ventricular meningitis.

MENINGITIS SECONDARY TO PHLEBITIS OF ARACHNOID VESSELS

Meningitis that develops during a hematogenous infection of the mastoid or of the nasal accessory sinuses is generally of vascular origin. The infection enters the meninges by an osteothrombosis of the small vessels; after it has passed through the dura, the cortical intracerebral veins are involved. The infection now spreads in the walls or within the lumen of the veins, which, being without fibrous covering, break down,

10. Eagleton, Wells P.: Surgical Treatment of Meningitis: Areas of System Primarily Attacked, J. A. M. A. **83**:1900 (Dec. 13) 1924.

with the formation of local abscesses in the arachnoid or in the subdural space; the ulcerating vein disappears in the pus.[11] In my experience, such local abscesses frequently are adjacent to the petrous pyramid, especially in the region of the saccus endolymphaticus.[12]

On several occasions the operative finding of such an abscess has led me to regard the local abscess as the cause of the meningitis, a mistake that would have been avoided had I appreciated the significance of the positive blood culture without venous sinus thrombosis.

Meningitis secondary to phlebitis of the arachnoid vessels rarely remains confined to an arachnoid cistern; it rapidly travels over both the base and the cortex. If the infection reaches the veins within the chiasmal cistern, the infected fluid is at once disseminated to all parts of the cerebrospinal system, because of the free communications that this large mesial cistern has with the other cisterns of the base. This is strikingly seen in fulminating meningitis from facial infection which retrogrades into the inferior cerebral veins.[13]

DIFFERENTIAL DIAGNOSIS OF THE ORIGIN OF A BLOOD STREAM INFECTION IN MENINGITIS

Immediately on the appearance of meningeal symptoms, it must be determined whether or not a blood stream infection is present; and if so, to diagnose if the disease is within a venous sinus, i. e., (*a*) sinus thrombosis, or is an infection of the small veins of the bone, i. e., (*b*) osteothrombophlebitis, or has attacked the arachnoid veins, i. e., (*c*) phlebitis of the intracerebral vessels. For when the infection is within a large sinus or is confined to the small veins of the bone, it is controllable surgically. But if the phlebitis has passed into the vessel of the arachnoid, intradural surgical intervention can be of little value, although treatment by repeated transfusions, especially from immunized donors,[14] offers some hope.

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11. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases, Edinburgh, Oliver & Boyd, 1931; Study of Paths of Infection to Brain, Meninges and Venous Blood Sinuses from Neighboring Peripheral Foci of Inflammation: Acute Infection of Left Sphenoidal Air Sinus, J. Laryng. & Otol. **42**:184 (March) 1927.

12. Eagleton, Wells P.: The Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases, J. Laryng. & Otol. **44**:657 (Oct.); 721 (Nov.) 1929. "Venous infection of the arachnoid mesh from an adjacent infection in petro-temporal and fronto-ethmo-sphenoidal regions."

13. Eagleton, Wells P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1926, p. 29. Case 2. Fulminating cavernous sinus thrombosis from stye in eye-lid with slight symptoms until twelve hours before death.

14. Eagleton (footnote 10, p. 1905).

INTRACISTERNAL AND INTRACAROTID INJECTIONS OF SERUMS AND DRUGS

Injections of serums and antiseptics into the arachnoid cavity [15] or into the carotid,[16] in my experience, have failed to be beneficial, and have been followed by disastrous results in several instances.

ENCEPHALITIS IN MENINGITIS FROM INVOLVEMENT OF THE VENOUS SYSTEM OF THE VENTRICLES

In many of my cases in which there is a positive blood culture, the operative investigation of neighboring venous sinuses revealed no evidence of thrombophlebitis, and drainage of a basal cistern failed to check the septic meningitis. These cases were accompanied by signs of cerebral compression. The patients did not have the usual symptoms of cortical meningitis, in that they did not complain of headache; they were not restless, and they did not scream, the outstanding symptom being a drowsy condition from which they could be aroused. This drowsiness, although apparently from cerebral compression, was accompanied by a low grade papilledema, and in some cases there was an entire absence of changes in the nerve head.[17]

Pathology of Encephalitis from Blood Stream Meningitis.—Autopsies on patients who, during life, had the symptom complex of a basal meningitis, with semicoma and positive blood cultures, show characteristic lesions, the significance of which has not been previously appreciated. There is only a little exudate over the cortex, but all the basal cisterns are filled with fluid, and the arachnoid mesh over the anterior surface of the pons and that of the inferior and superior rings around the cerebellum are the seat of purulent exudate. The cerebral cortex is white, and the fissures are obliterated from compression.

In the interior of the brain there are (*a*) moderate distention of the lateral ventricles, (*b*) an edema of the choroid plexus with hemorrhages into the ependyma, (*c*) punctate hemorrhages into the cerebral substance, especially in the basal ganglions and in the pons and medulla, and (*d*) a general edematous condition of the cerebral tissue itself.

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15. Eagleton (footnote 12, p. 732).

16. Dearmin, R. M.: Meningitis of Otitic and Nasal Origin: Report of Two Cases of Pneumococcic Meningitis Treated by Intracarotid and Intracisternal Injections of Pneumococcic Antibody Solution and 1 Per Cent Optochin Hydrochloride, Thesis, Graduate School of Medicine, University of Pennsylvania, 1930.

17. This semicoma I described as an integral part of the syndrome of bulbar cisternal meningitis. (Eagleton: footnote 4.) This was a mistaken interpretation as, while this peculiar drowsiness does occur in bulbar meningitis, it results not from the meningitis, but from an encephalitis following infection of the system of ventricular filtration.

Venous Pathways into the Ventricular System from Mastoid Suppuration.—With the object of explaining anatomically how infection from the petrous pyramid may reach the ventricular system, dissections were made of the venous communication between the mastoid area and the mesencephalon in a number of brains in which the vessel had been injected.

Infected vessels which leave the temporal bone (chiefly the veins of the internal auditory meatus, the venules within the saccus endolymphaticus and those draining into the jugular bulb) invade the anastomotic basal vein of Rosenthal, retrograde thrombophlebitis of which will cause an encephalitis in the area draining its terminals. The encephalitis is associated with general edema of the brain and a nonobstructive type of internal hydrocephalus, causing cerebral compression. Rosenthal's vein collects blood from superficial terminals on the lateral and basal side of the cerebellum and from the cerebellopontile angle, as well as from the basal ganglions and the ventricular system drained by the veins of Galen. It passes through the cisterna ambiens surrounding the superior collar of the cerebellum. This cistern is always the seat of exudate in pial vessel meningitis of the posterior fossa. It is probably at this point that infection of Rosenthal's vein occurs in the terminal stages of subarachnoid space meningitis.

In addition, my dissections have shown the presence of an undescribed fair-sized vein, for which the name of vena cerebella lateralis is suggested. It communicates laterally with the superior petrosal sinus and mesially with Rosenthal's vein. Through this lateral vein, retrograde thrombophlebitis, which has originated from superior petrosal thrombosis secondary to mastoid disease or to suppuration of the petrous apex, may also reach the deep ventricular venous system.

THE SPHENOID AND PNEUMOCOCCIC MENINGITIS [18]

A series of autopsies has convinced me that when pneumococcic meningitis follows a cold in the head, the sphenoid should be suspected of containing the principal focus of infection which had ultimately resulted in the development of the meningitis, provided, of course, that no other infection, such as otitis or pneumonia, has been present *prior to the original headache.*

Surgical Anatomy of the Sphenoid Sinuses and the Spheno-Occipital Base.—The position of the sphenoidal sinuses and the unique mor-

18. (*a*) Weichselbaum, A.: Ueber seltener Localisationen des pneumonischen Virus, Wien. klin. Wchnschr. **1**:573, 595, 620, 642 and 659, 1888. (*b*) Ortmann, P., and Samter, E. O.: Beitrag zur Localisation des Diplococcus pneumoniae (Fraenkel), Virchows Arch. f. path. Anat. **120**:94, 1890.

phologic character of the bony spheno-occipital base are favorable to the development of bacteria and for their transmission directly into the pontile cistern.

The sphenoidal part of the base is the only spongy mass of cartilaginous blood-producing bone of the skull in which the blood spaces of bone marrow are in intimate relationship with the an air sinus, as the mucous lining of the sinus is separated from the marrow by only a thin and imperfect layer [19] of compact bone. In addition, perforating blood vessels normally pass from the submucosa of the sinus into the vascular spaces of the medullary substances.[20] On its cranial surface the endosteal layer of the spheno-occipital base is very thin. The dura covering the bulbar cistern is firmly adherent to it, while perforating vessels connect the medullary substance with the vessels within the arachnoid.

Vascular Transmission of Infection in Sphenoidal Suppuration.—Sphenoidal sinus suppuration may be associated with an infected thrombophlebitis of the small veins. In chronic disease of the submucosa of the sphenoidal sinus, the thin compact bony layer that lines the sinus is frequently absorbed, forming "bays." Through these defects, the result of chronic sphenoiditis, infected blood vessels communicate directly with the blood marrow space of the base.[21] In the absence of all macroscopic signs of bone disease, thrombophlebitis of the veins of the submucosa of the sphenoidal sinus may involve the blood spaces of the base, the infection traveling in the coat of veins [18b] which normally perforate the compact bone lining the sinus bone, or may pass into one of the venous plexuses which cover the spheno-occipital bone and which fill every crevice of the cranial base. Having reached the vascular marrow or the basal plexus, the posterior "perforating veins" may now carry the infection directly into the veins of the bulbar cistern. In all patients dying of basilar meningitis, this cistern invariably is the center of exudate.

COMPLICATIONS IN SPHENOIDAL SINUSITIS

Micro-organisms, which have developed in a specific type of tissue, when free in the circulation attack similar types of tissue in other parts of the body—a basic principle in immunity.[22]

19. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhange, Vienna, W. Braumüller, 1893.

20. Hajek: Pathology of the Accessory Sinuses, St. Louis, C. V. Mosby Company, 1928, vol. 2, p. 615.

21. Turner, A. L., and Reynolds, F. E.: Inflammatory Changes in the Sphenoidal Air Sinuses, J. Laryng. & Otol. **43**:37 (Jan.) 1928.

22. Frey, W.: Significance of Organs of Mesenchymal Origin for Capacity of Resistance of Organism Against Bacterial Infections During Periods of Growth, Klin. Wchnschr. **8**:1604 (Aug. 27) 1929; abstr., J. A. M. A. **93**:1513 (Nov. 9) 1929.

The veins of the sphenoidal sinus communicate freely with the venous sinuses of the dura—the cavernous and petrosal sinuses, and the basilar and carotid plexuses—as well as with the arachnoid vessels within the bulbar cistern. Through these communications bacterial emboli which have formed in the vessels of the pneumatizing sphenoid may reach any part of the return venous circulation of the head.

In cases of (*a*) thrombophlebitis of the vessels of the submucosa, the blood spaces of the base may be the seat of (*b*) local or extensive hematogenous osteomyelitis. The latter may exist for a long time without producing symptoms,[23] in which case an exacerbation of the sphenoid sinus suppuration may bring into activity (*c*) a latent osteothrombotic or an osteomyelitic process in the vessels of the sphenoid base.

Showers of Bacteria from the Sphenoid the Cause of an Embolic Otitis or Embolic Infection of Pial Vessels Adjacent to Temporal Bone.—In any of the foregoing pathologic processes, showers of bacterial emboli may be discharged into the circulation, which in turn may give rise to (1) embolic otitis with mastoiditis, or to (2) embolic infection of pial vessels adjacent to the temporal bone. When the bacterial infection enters a pial vessel, it results (*a*) in the formation of a local abscess within the arachnoid or (*b*) in the development of a general suppurative meningitis.

Elective Localizations[24] *and Pneumatization; Suggested Mechanical and Embryologic Explanation for the Selective Affinity of Pneumococci Type III Which Have Developed in the Vessels of the Pneumatic Sphenoidal Sinus, for Veins of the Pneumatized Temporal Bone.*—Embryologically, the sphenoidal base, the mastoid and the petrous bones develop together from the same type of hyaline cartilage, with the exception of that portion of the petrous bone which makes up the bony

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23. Thomson, St. Clair: Cerebral and Ophthalmic Complications in Sphenoidal Sinusitis, Brit. M. J. **2**:768, 1906.

24. (*a*) Rosenow, E. C.: Elective Localization of Bacteria in the Animal Body, in Jordan, E. O., and Falk, I. S.: The Newer Knowledge of Bacteriology and Immunology, Chicago, The University of Chicago Press, 1928, p. 583. (*b*) Cecil, R. L.; Nicholls, E. E., and Stainsby, W. J.: Bacteriology of Blood and Joints in Rheumatic Fever, J. Exper. Med. **50**:617 (Nov.) 1929; abstr., J. A. M. A. **93**:1763 (Nov. 30) 1929. (*c*) Hesse, W.: Experimentelle Untersuchungen zur Frage des Lokalisations-Vermögens von Bakterien, besonders im Hinblick auf tonsillogene sekundäre Erkrankungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:198 (July 3) 1930. (*d*) Meisser, J. G.: Further Studies on Elective Localization of Bacteria from Infected Teeth, J. Am. Dent. A. **12**:554, 1925. (*e*) Bernhardt, H.: Zur Frage der Fokalinfektion und der "elektiven Lokalisation," Ztschr. f. klin. Med. **117**:158, 1931. (*f*) Miller, A. A., and Bojarskaja, W. G.: Zur Frage der elektiven Lokalisierung der Mikroben, Ztschr. f. Immunitätsforsch. u. exper. Therap. **61**:4, 1929.

capsule of the labyrinth, the fetal matrix of which forms sesamoid bones in other parts of the body. The compact bony labyrinth capsule is never pneumatized. It secures its blood from fetally developed, intradurally placed vessels.[25]

On the other hand, the process of pneumatization of the mastoid and of the sphenoid begins after birth. Pneumatization occurs in clefts in those areas of the head where, during embryologic life, membranous (visceral mesodermal) and cartilaginous (paraxial mesodermal) bones are in apposition.[26] Pneumatization starts from clefts or cavities which have been formed during fetal life in the spongy substance of cartilaginous bones.[27] At birth these clefts or cavities are filled with an undifferentiated mesenchymal substance. Pneumatization is a specific type of childhood growth. Its object is the expansion of protective bony surfaces with the preservation of lightness. Growth by pneumatization is accompanied by the invagination of epithelium of the respiratory tract. Invagination is a common form of development during fetal life, but after the birth of the child it occurs only during pneumatization.

The ingrowth by the epithelium of the respiratory tract during bony expansion by pneumatization is accompanied by the formation of vessels that run in the submucosa.[28] These vessels all appear postfetally and arise from branches of the external carotid artery that do not enter the dura.

Thus in the sphenoidal bone, after the appearance of the sphenoid sinus (as the result of pneumatization), two vascular systems lie in apposition—intradural and extracerebral vessels—separated only by a thin layer of compact bony tissues. In sphenoiditis, as long as the inflammation remains confined to the extradural vessels of the sphenoid submucosa, the septic process can produce only local systemic disturbances, for all drainage, whether it is vascular or lymphatic, is entirely extracranial. When, however, the compact bony wall which lines the sinus is perforated either by infection of the vessels—thrombophlebitis—or by bony suppuration, caries or necrosis, the intracranial vascular system is invaded. Hematic drainage now takes place by way of venous channels that are within the dura; thus micro-organisms which have entered the intradural venule circulation from the sphenoidal

25. Bast, T. H.: Blood Supply of the Otic Capsule of a 150 mm. Human Foetus, Anat. Rec. **48**:141 (Jan.) 1931.

26. Fawcett, E.: Notes on the Development of the Human Sphenoid, J. Anat. & Phys. **44**:200, 1909-1910.

27. Rouvière and Rouvière: Sur le développement de l'antre mastoidien et les cellule mastoïdiennes, Bibliog. Anat. **20**:24, 1910-1911.

28. McMahon, Bernard J.: Histopathology of Sinusitis and Mastoiditis, Ann. Otol., Rhin. & Laryng. **38**:732 (Sept.) 1929.

region may pass to any part of the venous return from the brain or of its membranes, not only because of the absence of valves, but because the venous and fluid currents within the dura change their direction with alterations of pressure and with each respiratory impulse. Thus bacteria from the sphenoid, having once entered the cerebral or the arachnoid mesh venous system, may pass to the terminal veins, leaving the temporal bone—the veins of the saccus endolymphaticus, the internal auditory meatus and venules emptying into the jugular bulb—or the terminals of fetally developed vessels which have functioned in the development of the otitic capsule (the fibrous strands of which are evident throughout life). All of these areas apparently furnish favorable soil for the growth of pneumococci because of the "elective localization" [24a] that pneumococci type III that have developed in the venules of the sphenoid have for the temporal bone.

In caries or necrosis of the sphenoidal base, organisms rapidly invade all parts of the arachnoid mesh. My autopsies demonstrate that micro-organisms when free in the fluid have a tendency to lodge in the arachnoid prolongation surrounding the terminals of the sensory nerves, especially of the seventh and fifth.

The unique (1) vascular peculiarities of the sphenoid sinus—(*a*) the direct communication that diseased and infected extracerebral vessels have with intra-arachnoid vessels—mechanically account for (*b*) the free passage of bacteria into the drainage venules of the temporal bone. The invading bacteria possibly are attracted to the mastoid because of (2) the embryologic tissue affinities that exist in (*c*) the appositional development of the sphenoid and of the petrous bone, (*d*) the formation of air sinuses by the invaginations of respiratory epithelium and (*e*) the accompanying new postfetal vessels. These functional and embryologic similarities help in explaining the biochemical affinity that pneumococci type III which have developed in the blood vessels of the submucosa of the pneumatic sphenoid, have for similar vessels in, and adjacent to, air-filled spaces of the mastoid process and of the medullary substance of the petrous pyramid. For certain strains of micro-organisms, when free in the circulation, certainly have a biochemical affinity for mesenchymal structures that have evolved from embryologically allied tissue. This is seen clinically in cases of lateral sinus thrombophlebitis, in the uniformity with which attenuated hemolytic streptococci that have developed in the bone of the mastoid—a mesodermal cleft area—(but which micro-organisms have become attenuated in thrombi within the intima of the venous sinuses) lodge in the embryologically associated mesodermal cleft tissue of the synovial membrane [29] of the joints and the vessels of the neighbor-

29. Key, J. A.: Synovial Membrane of Joints, in Cowdry, E. V.: A Special Cytology, New York, Paul B. Hoeber, Inc., 1928, vol. 2, chap. 21, p. 737.

ing tissue. The mesenchymal fluid contents furnish an appropriate medium for bacterial growth.[30]

Similar functional and embryologic affinities may explain how infected emboli deposited in veins of the submucosa of the temporal bone do occasion (1) a peculiar type of embolic otitis and mastoiditis, or when lodged in the veins which leave the temporal bone give rise to (2) suppurative meningitis.

In the past, the outstanding symptoms of the secondary aural suppuration have caused the primary sphenoidal disease to be overlooked [31] and the resulting meningitis to be attributed to the ear.

Meningitis Apparently from Suppurative Otitis but in Reality Due to a Latent Sphenoiditis.—A positive blood culture appearing coincident with pneumococcic meningitis points to the sphenoid as the primary focus of infection, even if otitis is present, provided the blood stream infection is not the result of sinus thrombophlebitis. Autopsies have repeatedly revealed an unsuspected suppuration of a sphenoidal sinus or an osteomyelitis of the sphenoid base as the cause of the meningitis, the suppurative otitis or mastoiditis being but a concomitant infection secondary to the sphenoidal disease.[32]

A positive blood culture appearing coincident with pneumococcic or streptococcic meningitis without thrombophlebitis of a large venous sinus calls for a careful retaking of the early history of the illness to ascertain if the initial headache and vomiting did not occur *previous to all aural symptoms.*

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30. Eagleton, Wells P.: Infective Arthritis Complicating Otitis Media, Arch. Otol. **34**:483, 1905.

31. Of eight cases of meningitis of sphenoidal origin reported by Kramer, two showed an associated otitis. Kramer stated that in six of thirteen cases of meningitis from the sphenoid, collected by Berger, an existing otitis was mistaken to be the cause of the meningitis. Kramer, R.: Intracranial Complications Following Sphenoidal Infection, Laryngoscope **39**:573 (Sept.) 1929.

Also Fremel's case of streptococcic meningitis was primarily from the sphenoid, as the patient had a chill, with a normal lateral venous sinus, a spontaneous rupture and contralateral abductus paralysis. "The ear was very little affected." Fremel, F.: Ueber Meningitis nach Keilbeinhohlen- und Mittelohreiterung, Monatschr. f. Ohrenh. **4**:45 (Jan.) 1922.

32. Culture in Lemere's case yielded a gram-positive bacillus (diphtheroid organisms); later culture showed a small gram-negative bacillus, most probably of the proteus variety; there were also a few gram-positive diplococci that looked like pneumococcus. Death occurred from meningitis. Postmortem examination showed the sphenoid sinus filled with pus. There was necrosis of the apex of the right temporal bone. The process was confined to the medullary bone, the mastoid cells being free. Lemere, like all others, failed to appreciate the significance of the sphenoidal suppuration, and of the embolic character and also that the primary infection was an acute hematogenous osteomyelitis of the sphenoidal base. Lemere, H. B.: Progressive Osteomyelitis of the Frontal Bone, J. A. M. A. **80**: 596 (March 3) 1923.

Frequency of Sphenoidal Suppuration in Meningitis Apparently of Aural Origin.—Fifty-eight autopsies in cases of meningitis in which there is a record of the condition of the sphenoid and in all of which the meningitis during life was thought to be of aural origin showed disease of the sphenoid sinus in 25 (or over 40 per cent).

This undoubtedly does not represent a proper proportion of cases of sphenoidal suppuration in relation to the total number of cases of meningitis, as there were 105 autopsies. It suggests, however, that (1) a considerable percentage of all cases of meningitis are of pial vessel origin, and among the latter, (2) those of pneumococcic type III origin, a large proportion, if not all, have their primary focus in the sphenoid.[33]

CLINICAL COURSE OF EMBOLIC OTITIS, THE RESULT OF SHOWERS OF BACTERIA FROM INFECTED SPHENOID VESSELS, WHICH TERMINATES IN PNEUMOCOCCIC MENINGITIS

The patient at first suffers from a "cold in the head" with more pain than that usually accompanying a rhinitis.

1. *Stage of Primary Thrombophlebitis Within the Mucosa of the Sphenoid.*—The initial pain is apt to be attributed to "sinus infection from la grippe." The pain, however, is not definitely over the frontal sinus, as is the pain from frontal sinus disease, but is a headache situated slightly above the sinus in the frontal region of the head, and, moreover, it is associated with nausea and possibly vomiting.

Even during this early period, a possible cerebral complication should be considered because of the headache and vomiting.

2. *Stage of Discharge of Embolic Bacterial Foci from the Sphenoidal Vessels into the Temporal Bone.*—The otitis is characterized by spontaneous rupture of the drum membrane with little or no pain.

The drum membrane in patients examined before the aural discharge was found to be reddened in the upper quadrant, without bulging. The spontaneous rupture is the first clinical manifestation of the stage of bacterial infarcts into the vessels of the temporal bone. Pain in the ear, when present at all, never has the intense bursting character of acute suppurative otitis media. Several of my patients had no pain prior to the spontaneous rupture. One boy had no intimation whatsoever of any aural disturbance; on awakening, he surprisedly said, pointing to the discharge on his pillow, "Look at what came out of my ear in the night."

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33. Since presenting the foregoing data, I know of four other cases of pneumococcic type III meningitis that apparently came from the ear, in which the autopsy showed an infection of the sphenoid.

Nutritional Death the Cause of the Spontaneous Rupture and of the Aural Discharge: Ordinary acute suppurative otitis media in which the infection ascends from the nasopharynx by direct extension through the eustachian tube gives rise to an exudate within the cavity of the middle ear, with distention of the drum, which produces severe pain before its rupture.

In embolic otitis, a shower of the bacteria from the sphenoid enters the circulation, passes through the squamopetrosal suture and lodges in the small vessels in the superior part of the drum membrane and of the middle ear, which anastomose with vessels that perforate the suture. The bacterial occlusion occasions nutritional death of the tissue of the drum membrane and of the middle ear, with a purulent aural discharge.

If the bacterial shower does not cause bone destruction, the aural process soon heals. This doubtless is the usual termination, as sphenoidal sinus thrombophlebitis must be controlled by nature's protective process, even after the discharge of a "shower" of emboli, in the vast majority of cases.

In several of my cases the aural discharge stopped almost immediately, leaving only a "full feeling" with diminished hearing. This was associated with a lusterless appearance of the drum, such as is frequently seen in pneumococcic type III otitis (the old Streptococcus mucosus-capsulatis otitis). When there is bony destruction in a part which communicates with the cavity of the middle ear, the discharge is profuse.

For a time the otitis apparently runs the mildly febrile course of an ordinary suppurative otitis media, with the exception, however, that at irregular intervals there is pain in the head with attacks of nausea or vomiting. The vomiting is invariably disregarded as it is attributed by the patient to an indiscretion of diet or the taking of a medicament.

In most of my cases, the temperature, vomiting and cephalalgia at times subsided but returned. Several patients were subjected to a paracentesis of the drum in spite of a free aural discharge. In the cases in which healing had occurred the drum was inflated in an effort to relieve the "full feeling."

The pain in the head is apt to be troublesome only at night. It is usually referred above the ear as in mastoid suppuration, the symptoms of which now completely mask the primary lesion in the sphenoid.

3. *During the State of Semiquiescence of the Sphenoidal and Otitic Infection.*—The roentgenogram in several of my cases revealed a moderate cloudiness of the cellular structure of the mastoid, which in conjunction with the pain and the aural discharge led to an operation on the mastoid.

In embolic pneumococcic type III otitis, the semiquiescent stage continues, as a rule, about seven days, although in one of my cases it

lasted nine weeks. During the entire time there was no discharge from the ear, although the patient had intermittent attacks of frontal or temporal headache and a slight but irregular temperature.

4. *Repeated "Showers" of Infected Bacteria.*—In the semiquiescent period repeated showers of infected bacteria may be poured from the infected pneumatizing sphenoid into the circulation. In pneumococcic type III infection, the bacterial showers certainly have a predilection for the veins connecting the meninx with the temporal bone, but bacteria may lodge within the vessels of the cerebral substance.

Thus several patients had attacks of labyrinthine vertigo and nystagmus; one had sudden total deafness with facial paralysis without vertigo or dizziness, suggesting a postlabyrinthine involvement. Several had involvement of the opposite ear, characterized by deafness without pain.

A slight *preliminary shower* may be lodged in the cerebral substance, as I have seen a sudden loss of accommodation associated with a dilated pupil, probably as the result of a small infarct. Both symptoms disappeared in a few days.

Invasion of Meninges.—In the cases of embolic pneumococcic type III otitis that progress to suppurative meningitis, the acuteness of the onset of the meningitis will depend on whether the final overpowering shower of bacteria finds lodgment (*a*) within the capsule of the petrous bone (from which the infection extends into the arachnoid), in which case invasion of the meningitis is delayed, or lodges (*b*) within a pial or a deep cerebral vessel, when the meningeal onset may be almost apoplectic in character.

Anatomic Location of Embolic "Showers" from Vessels of the Pneumatizing Sphenoid, Which Lodge in Vessels in the Morphogenetic Pneumatized Temporal Bone.—I have had cases in which embolic showers invaded (*a*) the veins which run along the superior surface of the petrous pyramid, and (*b*) the veins which are within the medullary substance of the petrous apex. In both cases the patients had marked signs of meningeal irritation, which, however, rapidly disappeared.

Infection of Pial Vessels Connecting the Petrous Portion of the Bone with the Brain.—On the other hand, I have seen patients who presented symptoms that warranted the belief that a shower of bacteria had lodged (*a*) in the veins of the saccus endolymphaticus, (*b*) within cerebral veins which empty into the jugular bulb and (*c*) in the veins which pass through the internal auditory meatus. All of these cases had outstanding meningeal symptoms, which, however, subsided, more or less for a time, because in these areas the arachnoid cells have the power to limit suppuration and to control infection.

Immediate Embolic Invasion of the Deep Cerebral Vessels and the Venous System of Galen.—In other instances a shower involved the deep ventricular system of the brain with signs of encephalitis; and in one instance a "shower" probably lodged in vessels of the bulbar region, as it was associated with sudden unconsciousness and embarrassed respiration followed by the almost immediate death of the patient.

5. *Stage of Acute Meningeal Extension with an Immediate Blood Stream Infection.*—Usually, after a surgical trauma such as a mastoid operation—in one case after an inflation of the ear—or following an intercurrent infection such as a tonsillitis, any of which by lowering the resistance may cause a recrudescence of a latent infection in the sphenoid, there suddenly occurs a chill or chilly feelings followed by a sharp rise of temperature with a severe headache and vomiting.

In contradistinction to subarachnoid space meningitis, the blood culture in meningitis from embolic invasion of the pial vessels is immediately positive. That the blood stream infection is not from a thrombus within a large venous sinus is shown by the fact that in my cases the operative investigation of the lateral sinus and jugular bulb gave uniformly negative results and the postmortem examinations failed to disclose any infection in them.

With the onset of meningitis, the leukocytosis is very high. In the fulminating cases the cerebrospinal fluid, while clear, contains pneumococci; in the cases that originate from osteomyelitis of the sphenoidal basis or of the petrous apex, the invading organism generally is a streptococcus.

Headache and Cerebral Compression in Meningitis Secondary to Phlebitis of Arachnoid Vessels, with Embolic Otitis from the Sphenoid.—If the infection is limited to the basal cisterns, there is little or no headache after the period of invasion. If the cortex is involved, headache is an outstanding symptom.

In the basal cases, encephalitis and edema of the brain, with a nonobstructive internal hydrocephalis associated with signs of moderate cerebral compression, develop early. In the milder types the patient survives several days or even as long as two weeks. In the fulminating cases death may occur within a few hours, in which event the patient dies from an overpowering cerebral toxemia, the postmortem examination showing multiple hemorrhages into the cerebral cortex without bacteria, although the fluid in both the arachnoid and the sphenoid is laden with micro-organisms.

A child was thought to have measles because coryza was accompanied by a purplish rash with headache and vomiting. All symptoms soon disappeared. A few days later there occurred a sudden rise of temperature with bilateral deafness

without pain in the ears. There was spontaneous rupture of one drum and paracentesis of the other, as it was found to be reddened, followed by a profuse discharge from both ears. Slight tenderness was present over one mastoid. Double mastoidectomy was performed. Both mastoids were found to be hemorrhagic, the one without tenderness being the most involved. On the second postoperative day there was a steeplechase temperature with a positive blood culture. The sigmoid sinus was investigated and found unaffected. Early appearance of symptoms of basilar meningitis was present, but without headache. The basal cistern was drained. There was cerebral compression due to edema (from obstruction of the deep veins draining into the veins of Galen). Until death, the patient lay on his back with his eyes closed, in a varying degree of coma, with a stiff neck and slight papilledema.

Postmortem examination showed the cortex compressed; a purulent basal cisternal meningitis and edema of the brain with moderate distention of the ventricles and punctate hemorrhages into the ependyma, pus in the sphenoid sinuses, with acute osteomyelitis of the sphenoidal roof.

Comment.—The sphenoidal suppuration undoubtedly began during the supposed measles, the existence of which is questionable, as the "plum colored" eruption was possibly a toxic erythema from the osteomyelitis of the sphenoidal roof. The meningeal involvement began at the time of the initial vomiting and headache.

The otitis and mastoiditis were secondary infections, the result of embolic showers from the accessory sinus, but gave rise to outstanding symptoms of aural suppuration, which overshadowed all evidences of the sphenoidal hematogenous osteomyelitis.

The (*a*) appearance of the headache and vomiting *prior to the aural infection,* in conjunction with (*b*) a positive blood stream infection occurring immediately with the appearance of meningeal symptoms, should have suggested the sphenoid as the primary focus.

CONCLUSION

1. There are two types of suppurative meningitis, depending on the mode of invasion of the infection and the method of its extension within the arachnoid, viz.:

(*a*) subarachnoid space meningitis and (*b*) meningitis secondary to infection of the pial vessels.

2. Subarachnoid space meningitis is curable surgically as long as the infection is limited to a basal cistern.

3. Meningitis due to infection of a pial vessel is uniformly fatal, at least when it has advanced beyond the neighborhood of the primary focus.

4. Pneumococcic type III meningitis secondary to the infection of pial vessels is generally of sphenoidal origin in my experience. In the acute cases, the route of the infection into the meninx is by blood vessels, the primary vascular lesion being microscopic; in the more protracted cases, the sphenoidal basis is the seat of a hematogenous osteomyelitis.

5. Consequently, the future surgical treatment of pneumococcic type III meningitis to be successful must be an early attack on the sphenoid.

The character of the operation will depend on whether the invading organism enters (*a*) from a thrombophlebitis of the submucosa or (*b*) from an osteomyelitis of the sphenoidal basis.

6. The theory is offered that pneumococci that develop in the vessels of the pneumatizing sphenoid have an selective affinity for the vessels of the pneumatized temporal bone, in which case a positive blood culture appears coincidently with the meningitis. Embryonal and mechanical facts in pneumatization suggest an explanation for the "elective localization" that certain strains of pneumococci which have developed in the sphenoid have for the temporal bone.

7. "Showers of bacteria" from the sphenoid sinus are the cause of a specific type of "embolic pneumococcic otitis," the secondary mastoiditis completely masking the primary inflammation in the sphenoid.

8. Embolic otitis is characterized by spontaneous rupture of the drum membrane without pain. A shower of bacteria from a thrombophlebitis of the sphenoid deposited in the veins adjacent to the petrous bone apparently is the cause of pneumococcic type III (Streptococcus mucosus-capsulatis) meningitis in a large proportion of cases.

9. A spontaneous rupture of the drum membrane with little or no pain is suggestive of a blood stream infection.

10. Streptococcus hemolyticus, which originates in blood-producing spaces of the sphenoidal basis or the petrous apex, also causes a meningitis of the pial vessel type. It likewise is associated with an immediate positive blood culture.

11. Streptococcus hemolyticus possibly may cause embolic otitis, but, in my experience, the meningitis from this organism is always the result of a localized collection of pus either in the mastoid, in a venous sinus, in an adjacent air space, or in the medullary substance of the sphenoidal basis or of the petrous apex. Consequently, from a surgical standpoint when meningitis from Streptococcus hemolyticus is associated with an immediately positive blood culture (and there is no sinus thrombosis and the mastoid has been well exenterated) an attempt to locate the primary purulent focus in the medullary substance of the petrous apex or the sphenoidal basis should be made.

12. Encephalitis as an early and frequent complication of meningitis secondary to infection of a pial vessel has been observed. Its cause is explained anatomically. The involvement of the ventricular venous drainage system is associated with edema of the brain and a nonobstructive internal hydrocephalus, which gives rise to a peculiar type of cerebral compression.

Clinical Notes

A METHOD FOR THE REPAIR OF A SYPHILITIC NOSE

C. R. Straatsma, M.D., New York

I have never been able to satisfy myself that the present methods for the repair of a syphilitic nose in which the lining has been destroyed are the best possible. There are two methods in common use: first, the removal of all scar tissue and the relining of the entire nasal cavity with a Thiersch graft, held in place by a mold, which in turn is attached to a denture previously applied; and second, the use of a full-thickness Wolfe graft. Both methods are uncertain, and as yet I have not seen a satisfactory outcome.

As I see it, the problem is to line the nose abundantly, so that there will be no subsequent contractures, and to support the nose with rib cartilage at a later date. Whatever lining is used, there must be sufficient room between the two layers (that is, lining and covering) for the insertion of a cartilage graft strong enough to support the nose. Even if the Thiersch graft has taken completely, I have found it impossible to make a tunnel between a thin Thiersch graft and the outer skin without perforating into the nasal cavity, thus exposing the cartilage or transplant to infection. If a full-thickness Wolfe graft would take in toto, so that it would be possible to tunnel for the later ridge support, it would begin to solve the problem, but I have failed to find a single instance in which this has occurred.

To overcome the objections to the foregoing methods and to repair at the same time those deformities that usually occur concurrently, such as collapse or partial destruction of the ala, absence or partial absence of the columella, fistulas anywhere in the nose or too short a nose, I have used a tubed pedicle graft from the arm.

Figures 2 and 3 are typical examples of noses that can be nicely repaired by the method which I am about to describe in detail.

Figure 4 shows a tubed pedicle flap that has been prepared on the inner aspect of the arm. The size of the pedicle is determined by the needs of the individual case, and a complete description of its preparation may be found in any textbook on plastic surgery. The distal end of the tubed pedicle is usually outlined, elevated and then sutured back into position on the arm. This is called the delaying of a tubed pedicle. This step is not absolutely necessary, but it insures the blood supply and lessens the chance of loss of any part of the tube.

Figure 1 attempts to show the procedure in detail. Figure 1 *A* shows a marked saddle nose, the outer covering of which is adherent to the remaining turbinate bones and septum. The columella is destroyed, except for a very narrow strip of

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From the plastic clinics of the New York Post-Graduate Hospital and Medical School and the Newark Eye and Ear Infirmary.

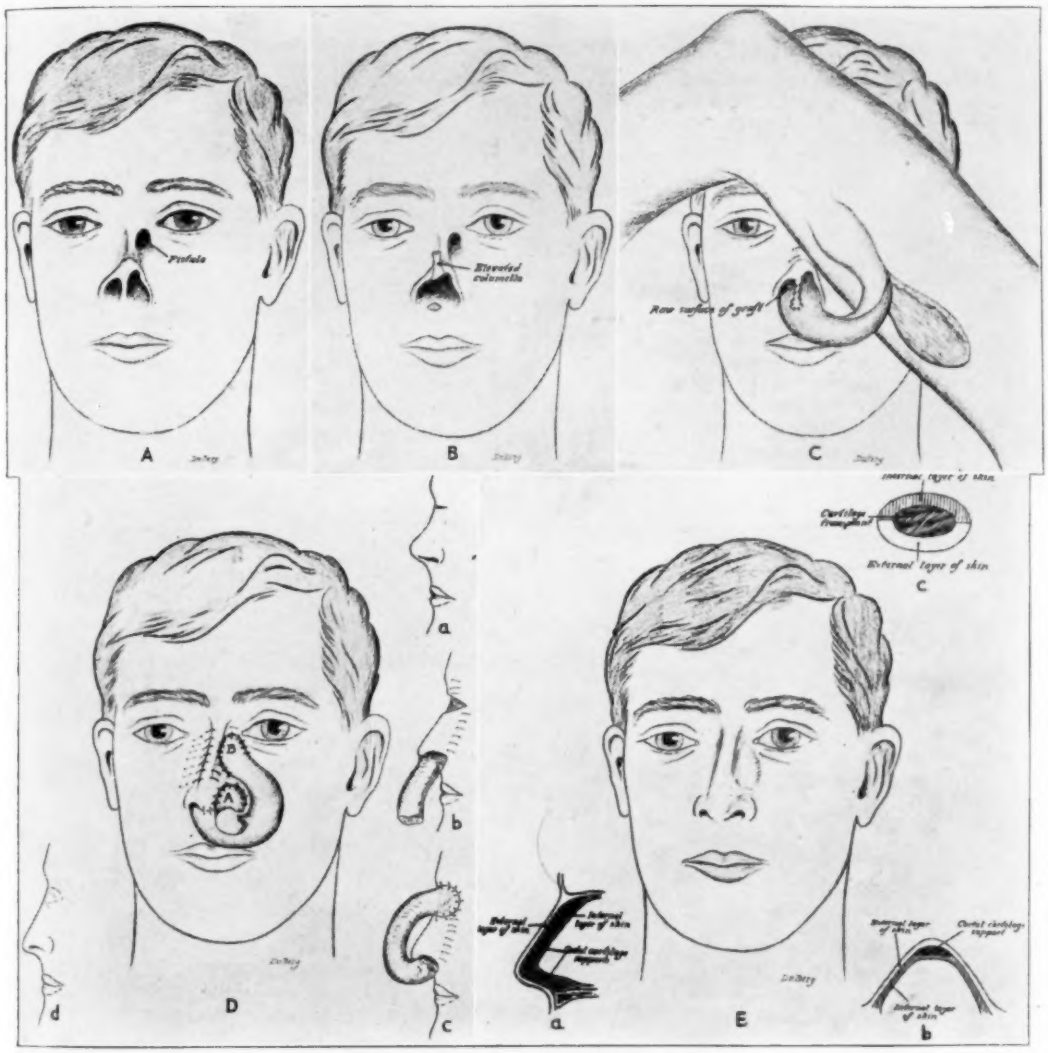

Fig. 1.—Details of the procedure described: *A*, saddle nose. *B*, severing and elevation of columella. *C*, tube planted inside the nose. *D*, repair of fistula. *E*, repaired nose.

Fig. 2.—Nose suitable for repair by method described.

Fig. 3.—Nose similar to that in figure 2.

Fig. 4.—Tubed pedicle flap on arm.

skin which is too small to admit a buttress support. There is also some destruction of the left ala and a fistula on the left side of the nose high up. To repair such a nose, the tubed pedicle, which has been previously prepared, is severed at the distal end, and if the blood supply is satisfactory, the nasal skin is then elevated. As can be seen in figure 1 *B*, the columella is severed at its base and elevated. The posterior surface of the columella is then freed from scar tissue to permit its attachment to the incoming flap. The skin of the bridge of the nose is then cut free from the remaining turbinate and septal structures, and all scar tissue removed from the inner surface of the nasal covering. This leaves an outer covering with an inner raw surface. Then to replace this destroyed lining, the tubed flap is inserted, the raw surface of the tube coming in contact with the raw surface of the nasal covering. To lengthen the nose, the scar tissue that causes its upward retraction is excised and the tip structures are placed in their proper positions before the insertion of the lining. This will leave, depending on the amount of lengthening, a portion of the lining exposed until the tube is severed from the arm; then the detached end is used to cover it. This is illustrated by figure 1 *D*, *A* to *D* inclusive. If there is complete loss of the columella, which occasionally occurs, this same tube can be used to replace it.

Figure 1 *C* shows the tube planted inside the nose. It is held in place by stitches and nasal packing. As can also be seen, the columella is sutured to the flap so that it can be made large enough and long enough to admit a cartilage buttress support. This tube is allowed to remain attached for two weeks, the arm being fixed meanwhile by a plaster cast or adhesive tape dressings.

Figure 1 *D* shows the severed end of the tubed pedicle swung up to repair the fistula, which has been previously described. As shown in *A* of the same figure, a portion of the flap may be used to repair any loss of either ala. After the nose has been relined and any other defects have been repaired, the rib cartilage ridge and buttress support can be inserted. This is done in the same manner as in repair of a saddle nose in which there has been no destruction of the lining. (A detailed description of this procedure can be found in "Plastic Surgery of the Nose" by Sheehan,[1] or in "Reconstructive Surgery of the Head and Neck" by Smith [2].)

Figure 1 *E* shows the repaired nose. Insert *a* is a longitudinal section of the nose, showing the cartilage planted between two full-thickness layers of skin. Insert *b* shows a cross-section of the nose with the cartilage in its bed between the two full-thickness layers of skin. Insert *c* is a cross-section of the columella, showing the layer of transplanted skin on its posterior surface and also the cartilage buttress support, which is completely encased by skin.

CONCLUSION

This method provides for complete relining of the nose as well as for the repair of any of the ordinary defects that occur in conjunction with syphilitic destruction of the nasal support, its lining or covering. The method has been used successfully in several cases, and I am offering it as a practical method for handling these surgical problems.

899 Park Avenue.

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1. Sheehan, J. Eastman: Plastic Surgery of the Nose, New York, Paul B. Hoeber, Inc., 1925.

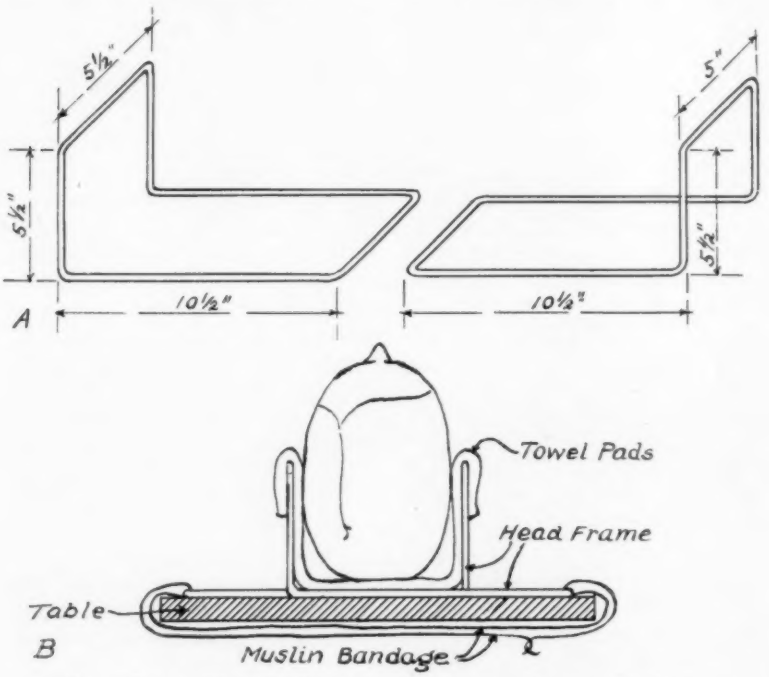
2. Smith, Ferris: Reconstructive Surgery of the Head and Neck, in Nelson's Loose-Leaf Living Surgery, New York, Thos. Nelson & Sons, 1928.

A FRAME FOR HOLDING THE HEAD IN OPERATIONS ON THE FRONTAL SINUS

LEE M. HURD, M.D., NEW YORK

By request I am reporting the simple frame that I have used for many years to hold the head firmly during the performance of radical operations on the frontal sinus with the patient under general anesthesia.

It consists of two halves that fit into each other. Each is made of round iron, one fourth of an inch (0.64 cm.) in diameter, of the form and dimensions given



Frame for holding the head in operations on the frontal sinus.

in *A* in the accompanying figure. They can easily be made by bending the stock while cold, with a bench vise and a heavy Stilson wrench, and the joint can be welded or brazed.

When in use, the head is held between the short arms of the two frames, which have been fitted together by sliding the long arm of the narrower frame within the long arm of the wider frame. Towels are placed to prevent undue pressure on the sides of the head and under the head to give the desired angle. The frame, and with it the head, is secured to the operating table by tying the long arms of the frame to the table with a long heavy muslin bandage which is knotted under or at one edge of the table (*B* in the accompanying figure). It is evident that

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the stronger the pull put on the ends of the bandage, the more firmly will the two parts of the frame be pulled together and the more tightly will the head be held.

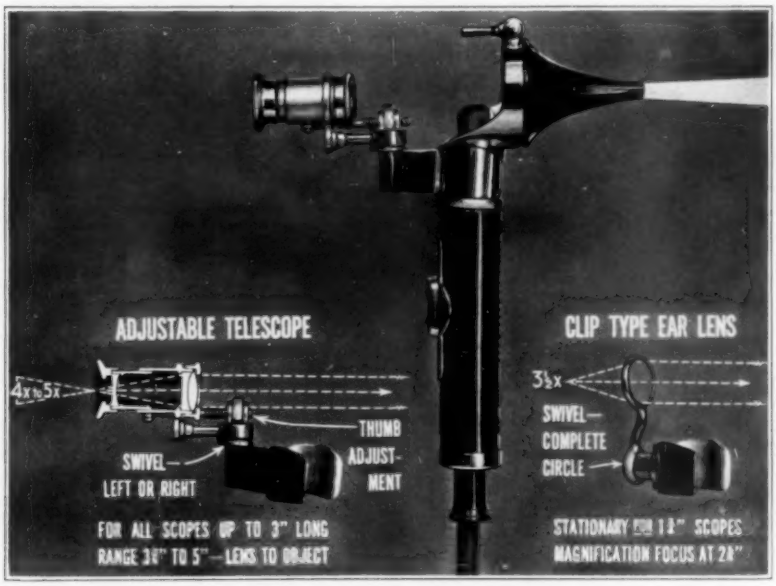
The entire frame is draped with sterile towels or a sheet, after the usual local preparation of the skin. The frame holds the head with all necessary firmness, prevents rotation and greatly aids during the use of the chisel and mallet and heavy bone rongeurs.

39 East Fiftieth Street.

AN ADJUSTABLE TELESCOPIC OTOSCOPE

M. Reese Guttman, M.D., Chicago

The older otologists, as is evidenced by their application of the lens system to the ear specula, appreciated the need of magnification in order to be better able to study the tympanic membrane and its varied pathologic manifestations. This



Adjustable telescopic otoscope.

same need was obvious at a later date with the introduction of the electrically illuminated otoscopes, which were almost always supplied with a lens system for magnification. The new electrically illuminated device with means of magnification has become very popular, especially with the general practitioner and the pediatrician. However, one drawback to its use is apparent: It has a lens system with a fixed focus. As is well known, the external auditory meatus varies in depth,

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depending on a number of factors, the most important being that of the age of the patient. A sharp image of the tympanic membrane could be obtained in the fixed focus instruments only by varying the depth of introduction of the speculum into the external auditory canal. Not infrequently, the bony external meatus proved a hindrance to the necessary deeper introduction of the speculum, and thus prevented accurate focusing with a resultant blurred image of the drum head.

In order to obviate this inherent drawback, an idea of an adjustable focusing device was conceived. Through the cooperation of the Cameron Surgical Specialty Company, a telescopic lens system that was adjustable was produced and perfected, and applied to their electrically illuminated otoscope. The accompanying diagram is self-explanatory. As may be noted from the illustration, the adjustable telescopic mechanism can be attached to the otoscope, when required, by means of a clip. This clip is connected by means of a swivel device to the lens system. The range and focus of the lens system are varied by means of a knurled knob, which is rotated until a sharp image of the object to be examined is obtained. The optical system has a range of about 6 cm., thus permitting sufficient latitude in viewing any variation of depth of the auditory meatus. The magnification obtained varies from 4 to 5 diameters. The swivel joint permits the use of a myringotomy knife or any of the instruments used for operation on the middle ear. In addition, a pneumatic chamber is supplied that can be used in conjunction with the telescopic system.

The same adjustable telescopic lens system is now being applied by Dr. Joseph C. Beck and myself, to an electrically illuminated combination instrument for the observation of the nasal chambers, postnasal space and larynx.

185 North Wabash Avenue.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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OTOSCLEROSIS

During the past year Guggenheim [1] wrote an illuminating article on the development of the stapes, in which he showed that the footplate of the stapes is really derived from the labyrinthine capsule and not from Reichert's cartilage as heretofore generally believed. In the human embryo of 5 weeks the labyrinthine capsule is composed of condensed mesenchyma and has no oval or round windows. At 7 weeks the precartilage is better formed and the capsule shows a deep indentation near the stapedial ring, but there is as yet no gap in the capsule. The perichondrium is plainly seen to be continuous over this area. At 9 weeks Reichert's cartilage forms a complete ring round the stapedial artery and indents the labyrinthine capsule, but the perichondrium is still unbroken under the ring. At 11 weeks the entire capsule is cartilaginous except at the internal auditory meatus and just beside the stapedial ring. In this area the capsular cartilage stops abruptly and the future footplate of the stapes is merely condensed mesenchyma. By this time the stapedial ring is pure cartilage, quite unlike the adjoining mesenchyma of the oval window. At this stage there is a condensation at each side of the ring indicating the position of the future annular ligament. At 13 weeks the segment of the ring next to the footplate has become flattened, but is of distinctly different structure from the footplate. At 16 weeks the annular ligament has almost its adult form. The stapes still shows a difference in texture between its two components. In the capsule itself on either side of the annular ligament the mesenchyma is condensed; this is the site of predilection for otoclerosis.

In the lowest vertebrates there are no labyrinth windows. In otosclerosis there is a process of closure of the oval window which, Gug-

1. Guggenheim, L. K.: Laryngoscope **41**:623, 1931.

genheim suggested, may be an atavism or a reversal to a primitive type rather than a disease process.

In another paper Guggenheim [2] gave a short résumé of the development of the organ of hearing, illustrated by photographs.

Fraser [3] described the condition of meatal atresia and maldevelopment of the middle ear. He traced the development of the various constituents of the middle ear from the bronchial arches and clefts. Microtia is due to some interference with development in the second month before the external meatus is formed. The auricle, malleus and incus are involved, all developing from the first and second arches. The labyrinth develops from the otic vesicle and is not usually involved in any developmental arrest associated with middle and external ear structure.

Microtia is not uncommon. It is more common in males than in females, and more common on the right side than on the left. Facial paresis and hemiatrophy are sometimes associated with it. The hearing defect is typically of the sound conduction type, and speech is usually heard. Operation is not advisable except in the case of infection.

Werner [4] has studied the growth process in the normal labyrinth capsule in its relationship to otosclerosis. In the human labyrinthine capsule there is little growth in extra-uterine life and none after the second year. Some growth takes place in the cartilaginous capsule up to the seventh month, when the capsule becomes completely surrounded by bone.

In certain areas of the labyrinth the endosteal lining is absent. This is seen on the convexity of the semicircular canals and at the apex of the cochlea, where it is the result of a process of growth. The semicircular canals grow slightly in size for a short time after birth and their angles change somewhat. In the growth of the canals there is resorption of the endosteum on the convexity of the curve accompanied by a corresponding deposition on the opposite side. At the point of greatest growth there is no endosteum, and the bone shows open spaces with blood vessels and cartilage cells lying free. This resorption seems to proceed in the absence of giant cells, as few are seen on the free surface execept occasionally in children, although they are common in the surrounding bone. The tip of the cochlea shows a similar denudation of endosteum, and the cartilage cells are in immediate contact with the cavity.

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2. Guggenheim, L. K.: Ann. Otol., Rhin. & Laryng. **40**:812, 1931.

3. Fraser, J. S.: Maldevelopments of the Auricle, External Acoustic Meatus and Middle Ear, Arch. Otolaryng. **13**:1 (Jan.) 1931.

4. Werner, C. F.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:128, 1931.

In the chicken the process of development of the labyrinth follows fairly closely that in the human being, allowing for differences in the shape and situation in the temporal bone. By the sixth month the bony changes are about complete around the bony capsule. There are areas with cartilage rests surrounded by more compact bone with cement lines where it has been laid down in layers. As in man, the enchondral bone at the periphery of the semicircular canals has been absorbed and may have disappeared entirely.

The normal development of the labyrinthine capsule in the chicken is finished in three quarters of a year at the most. The well known experiments of Wittmaack were all carried out on hens over 2 years old, so that the changes brought about by venous obstruction should not be mistaken for the normal large marrow spaces seen before growth is complete.

In the fowl there are some differences from the human labyrinthine capsule. The superficial layers of the capsule are changed into spongiosa and hence the capsule is thinner than in man. The venous network differs considerably from that in man, in whom the vascular areas remain unchanged through life and do not communicate with each other. An obstruction to the venous return in man would cause a circumscribed result, and hence otosclerotic foci remain localized. Otosclerosis occurs primarily only where the enchondral capsule with its original vessels remains. It does not occur at the convexity of the semicircular canals where the enchondral bone has disappeared. In experimental otosclerosis in the fowl the changes occur all over the capsule, but this is due to the different vascular arrangement.

Wittmaack,[5] ten years ago, came to the conclusion that ostosclerosis was due to venous obstruction. In his experiments on fowls he has repeatedly produced conditions that simulate closely those of otosclerosis.

The circulation of the blood in the temporal bone is not well understood, and the object of this paper is to show how venous stasis could occur. Sections of several temporal bones, including 3 from cases of otosclerosis, were studied. The vein leading from an otosclerotic focus is unusually large as compared with that from a normal bone. This is well shown in photomicrographs. When the vein that runs in the canalis nervi tympanici is followed upward, it is seen to leave the nerve and join the vein that lies in the facial canal. There is often a small twig which joins it from the dura mater. From then onward it joins the nervus petrosus superior major and enlarges and runs forward to join the plexus around the middle meningeal artery. In otosclerosis the large vein from the focus joins this vein near the facial nerve, after which it is much larger than normal.

5. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:150, 1931.

Wittmaack was unable to find any communication between these veins and the sinus petrous superior. The vein with the nervus petrosus superior major runs forward beyond the middle meningeal artery in many instances and joins the carotid plexus. In 2 of the 3 otosclerotic bones there was an unusually wide communication between this vein and the carotid plexus. In the other otosclerotic bone, in which the focus was small, there was no communication with the carotid plexus but only with the middle meningeal plexus. In 8 normal patients Wittmaack found this communication only once, and then it was very small.

These venous channels are relatively wide and form a network with wide spaces. Wittmaack thinks that it is quite possible that a reversed flow of the venous blood takes place at times. These veins are a sort of watershed between two circulatory areas. One goes forward, as described, while the other goes backward toward the sigmoid. Wittmaack thinks that a reversed flow of the venous blood probably has more to do with the otosclerotic changes than mere congestion. Incidentally, he attributed the redness of the promontory in otosclerosis to the venous congestion of the veins rather than to the bony changes themselves.

Wittmaack argued that if his theory was correct, that the congestion is brought about from back pressure from the carotid plexus and middle meningeal plexus, one should be able to stop it. He devised an operation to expose the middle meningeal artery and tie off the communicating veins. He suggested that this should be tried only in undoubted cases of otosclerosis with good pneumatization of the mastoid. If the mastoid is not well pneumatized, it is possible that there is an adhesive process present.

With regard to his experiments on hens, Wittmaack explained that hens were chosen because it was possible to produce venous congestion in them, while it was not possible anatomically to produce it in apes.

In another communication Wittmaack [6] also described his experimental work on hens. The results obtained experimentally are very similar to the changes observed in otosclerosis. The vessels are unusually large and numerous. The calcified zone that is normally present round the vessels is gone and there is an increase of cells round them. The bone is stained a deeper blue for some distance round the vessels. This blue bone shows an increase in the number of cavities and cells. Wittmaack stated that venous stasis is the obvious explanation of these changes, especially as they can be produced experimentally. In a further stage of the process the marrow spaces become larger until the picture resembles that of one of the stages of normal enchondral

6. Wittmaack: Pathogenesis of Otosclerosis, Arch. Otolaryng. **14**:186 (Aug.) 1931.

ossification. Still later there may be a gradual reossification of the perivascular spaces. The new bone differs from the older bone in that it shows no blue staining but is pink and laid down in irregular lamellae.

The fact that similar changes are not observed elsewhere is because of the unique structure of the labyrinth capsule and the peculiar distribution of the vessels with no anastomoses.

Dillon and Preobraschensky [7] have studied the relationship of otosclerosis to the pneumatization of the temporal bone. They examined the temporal bones in 50 cases of otosclerosis, or 100 bones in all. These were classed in three groups: (1) bones with well marked pneumatization, (2) bones that were pneumatic on one side and of mixed type on the other and (3) sclerotic bones. The authors concluded that in otosclerosis the degree of pneumatization is just as variable as in normal ears. The structure of the temporal bone has no obvious influence on the otosclerosis. The limited area of the capsular disease has no influence on the structure of the mastoid, as the development of the pneumatic cells is generally at an end before the otosclerotic process begins.

Weber,[8] investigating the subject of fibrils and cement lines in otosclerosis, used polarized light to clear up some of the points in dispute. With regard to the edge of the otosclerotic focus where the process stops abruptly with no visible cement line, polarized light reveals that the fibers stop abruptly and do not cross from the blue stained bone to the normal bone. Where the so-called "mantle formation" occurs, the fibers are seen to be curved, and in the hollow thus formed the deep blue bone lies. Weber thinks that otosclerosis is a new formation following previous absorption.

With reference to the bluish stain in otosclerosis, Hagens [9] stated that this has usually been attributed to the presence of calcium salts. These areas of blue staining with hematoxylin are especially marked around the small vessels. The author carried out some experiments on sections from 4 cases of otosclerosis to determine, if possible, what was the cause of the staining. Controls were made of other temporal and long bones.

Hematoxylin stains blue only when mordanted with salts of the heavy metals. The sections were first stained with a solution of aqueous hematoxylin. One gave a blue stain, the next was faintly stained and the other 2 did not stain at all. Alizarin and purpurin, which are used

7. Dillon, J. G., and Preobraschensky: Ztschr. f. Hals- Nasen- u. Ohrenh. **26**:537, 1930.

8. Weber, M.: Ztschr. f. Hals- Nasen- u. Ohrenh. **28**:415, 1931.

9. Hagens, E. W.: Otosclerosis: Staining and Chemical Reactions, Arch. Otolaryng. **13**:824 (June) 1931.

to detect the presence of calcium, were next used, with negative results. The presence of salts other than calcium was then studied.

The sections were stained with ammonium sulphide. A green color resulted in the foci in one case, but not in the others. This chemical reaction indicated the presence of iron in this specimen. The sections were then treated with an acid solution of potassium ferrocyanide, and the typical prussian blue reaction resulted. The foci of the 2 bones which stained blue with hematoxylin stained blue with this reaction. Various modifications of the foregoing tests confirmed the presence of fixed iron in the otosclerotic foci.

Mayer [10] has published a "Polemik" answering Brühl's objections to his theory of spontaneous fractures of the labyrinth. He affirmed that the fractures which he described were not artefacts but had occurred intra vitam, as revealed by the presence of repair tissue in them. (This is denied by many of his critics.) He has found fissures in the endosteal and enchondral bone at the site of predilection for otosclerotic foci. According to his argument, otosclerosis develops there because that part of the bone is subjected to excessive strain. In otosclerosis the process consists in replacement of finished bone by immature fiber bone. He drew a comparison with the so-called "Looser Umbauzonen" (zones of transformation in which the lamellar bone disintegrates and fibrous marrow is formed) which serve to produce a resisting bone at situations in the bone where there is irritation or strain.

Leiri [11] expressed much the same views as Mayer. He also believed that certain parts of the labyrinth are subjected to severe mechanical strain and that otosclerosis is a tissue reaction to this. He compared the appearance of otosclerosis with that of certain diseases of the epiphyses in children known as osteochondritis deformans juvenilis, which is said to be caused by undue strain. He believed that in both cases strain is an important factor superadded to some unknown predisposing cause.

Gale and Jaffe [12] published a short note on hyperparathyroidism in relation to the labyrinthine capsule of the guinea-pig. Hyperparathyroidism produces generalized decalcification of the skeleton. Experimental parathyroidism in guinea-pigs had no effect on the labyrinthine capsule, although the surrounding bone showed changes. The authors attributed this to the fact that the labyrinthine bone has a very low metabolic rate and hence is not susceptible to parathyroid extract.

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10. Mayer, O.: Ztschr. f. Hals- Nasen- u. Ohrenh. **28**:228, 1931.

11. Leiri, F.: Acta oto-laryng. **16**:57, 1931.

12. Gale, C. K., and Jaffe, H. L.: Labyrinthine Capsule in Guinea-Pigs with Hyperparathyroidism, Arch. Otolaryng. **14**:815 (Dec.) 1931.

Warwick,[13] writing on otosclerosis, stated that he preferred to call it otitic senility, but did not give any reason for this somewhat unusual idea. The gist of his speculation was that otosclerosis starts in fetal life or infancy and is due to a deficiency of vitamins A and D in the diet of the mother and the infant.

Duerto[14] reported on Sourdille's operative treatment of otosclerosis. The immediate results are good, but it is too early to be sure of the ultimate results.

CATARRH OF THE MIDDLE EAR

Richter[15] has made a study of the relationship of disease of the nose to that of the ear in a large series of patients in the University Klinik of Erlangen. Shortly condensed, his comments were: Acute infectious rhinitis is the commonest cause of tubal closure and otitis media. Vasomotor rhinitis, a deviated septum and ozena have little to do with the causation of otitis media. Inflammation or infection of the paranasal sinuses, on the other hand, is a fruitful source of aural infections. A deviated septum, vasomotor rhinitis and chronic hyperplastic rhinitis have been greatly overrated as causes of tubal closure. In 42 of 47 cases of chronic rhinitis without obvious disease of the nasopharynx both ear-drums were normal.

Richter condemned indiscriminate operations in cases of otosclerosis. They are usually performed on account of a wrong diagnosis and with an erroneous idea of the etiology, and have not the slightest result on the condition of the ear.

Smith[16] studied the basal metabolic rate in 46 cases of catarrhal otitis media. No cases of otosclerosis were included. Of these 46 cases, 22 gave normal readings; in 4 the values were above normal (+ 10 or over) and in 18 below — 10, while 2 gave doubtful readings. In the 18 cases showing a subnormal rate hypothyroidism predominated.

The symptoms could be classified into three groups: (1) neurasthenia, exhaustion, etc.; (2) inflammation of the mucous membranes, colds, etc., and (3) deafness and tinnitus only.

Glandular therapy produced marked improvement in those early cases in which it was indicated. On the basis of his work, Smith advised checking of the basal metabolic rate in all cases of catarrhal otitis media.

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13. Warwick, H. L.: Laryngoscope **41**:757, 1931.

14. Duerto, J.: Rev. españ. y am. de laryng., June, 1930.

15. Richter, H.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:30, 1931.

16. Smith, C. S.: Basal Metabolism in Middle Ear Catarrh, J. A. M. A. **97**: 132 (July 11) 1931.

Retraction of the drum membrane with loss of hearing for low tones was attributed to spasm of the tensor tympani by Josephson.[17] A common finding is a retracted ear-drum with a patent tube. The spasm is the result of an exaggeration of the normal protective mechanism. The tensor tympani and the drum membrane probably have as one of their functions that of protecting the ear from loud noises. In some of these cases there is an improvement of hearing after myringotomy. Both the deafness and the retraction of the drum are relieved by galvanism.

INNER EAR

An interesting report by Fabinyi [18] on the inner ear in arteriosclerosis has appeared. The report was based on the study of the microscopic sections and the functional results of hearing tests in 26 cases of arteriosclerosis. The clinical diagnosis of arteriosclerosis was confirmed in all cases. No cases of syphilis or otitis media were included. The ages ranged from 47 to 78, the majority of the patients being over 60. Sclerosis of the large arteries and of the smaller cerebral arteries was found, and the blood pressure was raised in the majority of the cases. The results of Fabinyi tallied closely with those of Bunch and Raiford, who studied the loss of hearing in advancing age, but the deafness was a little more marked.

The changes in the lower basal coil of the cochlea were more marked in the spiral ganglion and nerve fibers than in the organ of Corti. Atrophy of the organ of Corti in the lower basal coil was observed 7 times (27 per cent), though in 4 instances on only one side. In 4 cases there were definite changes in the vessels of the internal auditory meatus. Definite atrophy of the ganglion cells was present in 17 cases (65.4 per cent). In some areas the nerve fibers in the spinal lamina were absent.

The changes in the arteries of the ear were not like those of the larger vessels. The alterations consisted of proliferation of the intima and subendothelial tissue, hypertrophy of the internal elastic lamina and thickening of the tunica media. These changes were found in 57.7 per cent. In the majority of the patients the loss of hearing was greater than the average for their age. In most of the cases there was hypertension.

An unusual case was reported by Söderberg.[19] The case was one of absence of vestibular function with normal hearing, which the author believed to be unique, as he could find no record of any similar condition. The patient was a man aged 42 who had had a healthy child-

17. Josephson, E. M.: Spasm of Auditory Accommodative Mechanism, Arch. Otolaryng. **13**:166 (Feb.) 1931.

18. Fabinyi, G.: Laryngoscope **41**:663, 1931.

19. Söderberg, F.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:335, 1931.

hood. Erythema nodosum and epidemic parotitis occurred at the age of 20. He had severe headache and vomiting with the parotitis. Since childhood he had had difficulty in walking in the dark and in balancing. He had been subject to headaches brought on by cooling of the head. On examination the general condition was good. There was some spontaneous horizontal and rotary nystagmus to the right, increased on looking to the right, with slight nystagmus to the left. Romberg's test gave uncertain results, with a good deal of swaying. The gait with the eyes shut was uncertain and ataxic. Vestibular responses were entirely negative to caloric, rotary or galvanic stimuli. There was no change in the nystagmus after syringing. The patient could walk immediately after rapid rotation. The spontaneous pointing test gave normal results. There was no change after rotation. The hearing on both sides was normal, as was localization of sound. Both ear-drums, the nervous system, internal organs and blood were normal. The Wassermann test of the blood was negative. Roentgenograms of the ears and cranium were normal. Söderberg believed that the nystagmus was central in origin.

Hart [20] reported a case of complete bilateral deafness and almost total absence of vestibular response in a man aged 30 who had been taking 20 grains (1.29 Gm.) of acetylsalicylic acid a day for six weeks. After the onset of the deafness he took cinchophen, 5 grains (0.32 Gm.) every four hours, to a total dosage of 120 grains (7.77 Gm.).

In the same paper he described a case of bilateral deafness which he believed to be due to an alcoholic neuritis of the eighth nerve. A man aged 25 had contracted syphilis several months previously and had had some treatment, which was later repeated. On examination the Wassermann reaction of the blood and spinal fluid was negative and the spinal cell count normal. The patient had been drinking heavily for years, and in the last three weeks had consumed a quart of "sugar" liquor daily. Hart felt justified in attributing the deafness to alcohol rather than to syphilis on account of the negative Wassermann reaction, which is usually positive if there is any active disease of the central nervous system. The patient had also had prompt antisyphilitic treatment ten months before the onset of the deafness. Both the galvanic and the caloric reactions were negative.

A case of total bilateral loss of labyrinthine function, probably due to hemorrhage, was described by Rosenbluth.[21] A man had contracted syphilis ten years previously. Six weeks before his admission to the hospital he had had some injections of arsphenamine. Since then he had become pale and weak and had had numerous hemorrhages from the

20. Hart, V. K.: Laryngoscope **41**:557, 1931.

21. Rosenbluth, M.: Laryngoscope **41**:821, 1931.

nose. He was admitted to the hospital for severe epistaxis. The Wassermann reaction was negative on admission. He required repeated transfusions. There were hemorrhages in the retina and a submucous hemorrhage in the palate. Several days later he had a double earache with bulging drums, which ruptured and discharged blood. Both mastoids were tender. Some days later the patient became suddenly stone deaf. A prolonged caloric test was negative on both sides. There was no nystagmus. The rotation test carried out some time later produced no response.

A few days after admission arsenic was found in the stool. It disappeared two weeks later. The loss of hearing occurred twenty-four days after this, and the author was therefore of the opinion that the deafness was not due to arsenical poisoning directly, although the aplastic anemia probably was.

Wittmaack [22] has written another article on sound damage to the inner ear in answer to some references to his work by Barth. Barth had doubted the possibility of damage to the organ of Corti by vibrations transmitted from the floor through the bones. Wittmaack, in a characteristic fourteen page article, proceeded to show that Barth's and von Eicken's experiments were inconclusive and worthless because of faulty technic, etc. That these experiments gave negative results did not prove anything in relation to Wittmaack's carefully designed experiments.[23]

Losanov [24] studied the question of professional deafness in the nail factory at Saratov. The workers are subjected to continuous deafening noises from the stamping machines for many hours a day. His conclusions agree substantially with the observations of other workers in the same field, in that continuous loud noises affect the cochlea mainly. The perception of combined sounds is more affected than that of elementary ones. Long rest from work produces hardly any improvement. Cotton wool plugs do not protect the ear from such injury. Disease of the nasal accessory sinuses aggravates the condition. The author performed some experiments on animals which brought out nothing substantially new.

Katzschmann,[25] in an article on concussion deafness, stated that there is a recognized form of deafness among miners which has become more prevalent since the introduction of motor machinery in the mines. Ear tests in miners who had been using compressed air apparatus showed a reduction of hearing for a whisper of from 2 to 4 meters and an inability

22. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkophf. **130**:143, 1932.

23. Dickie, J. K. M.: Otosclerosis, Arch. Otolaryng. **1**:646 (June) 1925.

24. Losanov, N.: Acta oto-laryng. **14**:393, 1930.

25. Katzschmann: Ztschr. f. Laryng., Rhin. **20**:353, 1931.

to hear sibilants as compared with low tones. Bone conduction was diminished. Tinnitus was a marked symptom. Deafness and tinnitus made the workman much more liable to accidents. There was no vertigo or other vestibular symptoms. Katzschmann concluded that the condition was brought about by a combination of loud noise and mechanical vibration.

Krassnig,[26] in discussing the Ménière syndrome, pointed out that the theory of an angioneurotic disturbance does not explain satisfactorily all the symptoms. Thus, disturbances of hearing outlast the vestibular symptoms by days and even weeks, which does not fit in well with the theory of an angioneurotic disturbance. The cochlea is notoriously more susceptible to vascular changes, so why should the vestibular symptoms dominate the picture?

Migraine is quoted as a parallel disease. In migraine the attacks come on periodically, usually beginning at puberty and in females in association with menstruation. Ménière's syndrome is quite different. It affects people of from 25 to 30 or more years. The attacks come on with periods of catarrh. The scotoma of migraine affects healthy eyes or diseased ones equally, while Ménière's syndrome attacks only organically unsound ears. The scotomas in migraine last, at the most, only a few hours, while Ménière symptoms last for weeks.

Krassnig did not think that circulatory disturbances are the primary factor. Dederding had found changes in the auditory tube in most of her cases, and Krassnig agreed with this. He had made a study of 50 cases. Eighteen of the patients had organic disease from syphilis, meningitis, tumor or other causes. In the remaining 32 there was no definite anatomic change. All had one factor in common, namely, catarrh of the auditory tube. Treatment by catheterization of the tube brought about satisfactory cure or alleviation of the symptoms. The tensor tympani muscle must be involved in inflammation of the tube and probably undergoes contracture, altering the pressure in the labyrinth.

Krassnig concluded that the Ménière symptoms are due in part to tubal catarrh and impaired function of the tensor tympani muscle.

Summarizing a study of 135 patients with Ménière's disease, Mygind and Dederding [27] found headache in 91 per cent, generally localized to the side of the diseased ear. Tenderness of the mastoid process (Bárány's symptom) was found in seven cases. There was some relation between water metabolism and headache in many cases. Abnormalities in the cell count or other constituents of the cerebrospinal fluid were found in 90 per cent. The pressure of the fluid was generally

26. Krassnig, N.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **128**:141, 1931.

27. Mygind, S. H., and Dederding, D.: Acta oto-laryng. **14**:343, 1930

increased. Transient paresis of the abducens nerve was seen in 2 cases and of the facial nerve in 3 cases. The rare occurrence of medullary symptoms is against the aural phenomena originating in the medulla oblongata.

The cerebral, as well as the aural phenomena, would seem to be most rationally explained by the two unyielding liquor systems, the cranial and the labyrinthine cavity being affected by parallel processes consisting in an intracellular edema, which, as far as the brain is concerned, probably is principally localized in the meninges.

Proetz [28] discussed the various theories of the Ménière syndrome, most of which postulate vascular changes in the inner ear. He pointed out that allergy may account for many of these cases by producing either a localized edema or vascular disturbances, and he cited an interesting case illustrating such a condition.

A man aged 49 had had recurring attacks of vertigo, tinnitus and deafness lasting for variable periods. An elaborate physical examination gave substantially negative results. The patient was seen in one attack in which there was rotatory nystagmus to one side and horizontal nystagmus to the other side. One suspected tooth was extracted, and the patient was put on a diet of buttermilk and Bulgarian bacillus, whereupon the attacks became much worse. An edematous spot was seen in the nasopharynx and the question of allergy was raised. It was found that the patient was sensitive to milk, butter and cheese, which were thereupon eliminated from the diet. Since then the patient has been almost free from attacks. It is particularly interesting that he showed no cutaneous sensitiveness to milk products.

DEAFNESS IN GENERAL

Freund and Hays [29] reported 100 cases of deafness, but the value of the communication suffers considerably in that the cases were picked at random and conclusions drawn on the basis of a mixed group. The authors warned against excessive politzeration, as it may actually do harm. They stated: "A significant relationship between progressive deafness and faulty nose blowing was very apparent. Nearly every case that complained of ears puffing out when the nose was blown showed progressive diminution of hearing."

Emerson [30] contended that progressive deafness is a disease involving the perceptive apparatus, due in many instances to focal infection from a closed tonsillar crypt. He stated that concomitant changes in the tube and middle ear have no relation to the nerve dysfunction.

28. Proetz, A. W.: Ann. Otol., Rhin. & Laryng. **40**:67, 1931.
29. Freund, E. M., and Hays, H.: Laryngoscope **41**:326, 1931.
30. Emerson, F. P.: Ann. Otol., Rhin. & Laryng. **40**:9, 1931.

Coates,[31] in a paper on the prevention of deafness, described the manifold activities necessary to cope with this subject. It is estimated that in the United States there are about 3,000,000 deaf school children. Wide cooperation between otologists and public health authorities is necessary. Great progress has already been made in the control of deafness due to exanthemas, but there are other fields which require intensive study and cooperation between various bodies and, last but not least, sufficient funds.

Shambaugh[32] contributed a paper bearing on the same subject. There are about 45,000 deaf mutes in the United States. In a survey of 5,348 children sufficiently deaf to require segregation, three-fifths were congenitally deaf. In the etiology of the congenitally deaf, consanguinity and heredity are the most important factors. Of the patients with acquired deafness an enormous majority had become deaf by the fifth year. On this account it is necessary to concentrate attention on children of preschool age.

Meningitis is the commonest cause of acquired deafness, with measles second and scarlet fever and influenza next. Syphilis is not an important factor. Neuritis of the auditory nerve is usually responsible for extreme deafness following scarlet fever and measles. Many patients had no otitis media, and even in those who had that disease neuritis was more probably the cause than labyrinthitis, as the patients who had some vestige of hearing numbered more than twice as many as those who were totally deaf.

In the inspection of school children it is more important to have an otologist examine the ear-drum than to use elaborate audiometric apparatus, as what is needed is to check chronic diseases of the middle ear.

Otosclerosis cannot be prevented or cured by any known treatment, and Shambaugh warned emphatically against the many quack and semiethical treatments so much in vogue.

PATHOLOGY

Wittmaack,[33] in response to a criticism by Marx, expressed his views on the influence of inflammation of the middle ear in childhood.

He divided infantile otitis into three groups: (*a*) hyperplastic (*b*) exudative and (*c*) combined forms. The hyperplastic type is characterized by the formation of soft vascular organizing tissue with typical small vascular bridges connecting the organizing mass to the

31. Coates, G. M.: Ann. Otol., Rhin. & Laryng. **40**:651, 1931.

32. Shambaugh, G. E.: The Problems of Deafness, Arch. Otolaryng. **14**:36 (July) 1931.

33. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:207, 1931.

mucous membrane. The mucous membrane itself shows few changes, and there is an absence of small cell infiltration. Sometimes in the center of the exudate lying in the cavities of the middle ear, constituents from liquor amnii may be recognized. These masses probably come from the tube or the nasopharynx. The mucous membrane is purely myxomatous, and it is hardly possible that a severe inflammation could have been present and left no trace.

The exudative form is quite different. There is no organizing tissue. The mucous membrane is full of small cells extending deeply to the bone. There is a distinct tendency toward the formation of fibrils. Marx had suggested that both types were one and the same. Wittmaack denied this on the ground that the fibrous tissue infiltrated with round cells could never revert to the simple myxomatous type. The hyperplastic form is a foreign body reaction due to particles introduced during parturition and therefore sterile.

Wittmaack found combined forms frequently. The condition may be hyperplastic in the niches and exudative in the larger cavities. In mastoiditis in adults one finds cells with mild organization side by side with others in the most acute stage of inflammation. Wittmaack did not consider it conceivable that the mildly reacting cells had gone through the same stages as the severely inflamed ones. The inflamed cells may break into adjacent ones by erosion of the bone along the vascular channels or, on the other hand, may become gradually obliterated by new bone. This new bone has its trabeculae arranged radially and is divided by a cement line from the original bone. A few cells becoming obliterated in this way could not change a pneumatic mastoid into a solid one.

TUBERCULOSIS

Ormerod,[34] giving his experience of the last ten years in the Brompton Hospital, pointed out that tuberculosis of the ear is not common. Among 11,384 patients, about half of whom had tuberculosis of the lungs, only 58 or about 1 per cent of the phthisical patients, had tuberculosis of the middle ear. These figures do not compare with Logan Turner's and Fraser's, as theirs refer to patients of a different age period.

Ormerod believes that in the majority of the cases infection occurs through the auditory tube, but that bloodstream infection can occur, as infections of joints and bones sometimes occur at the same time as the aural involvement.

The onset is insidious and painless. Of 16 cases 6 started painlessly and discharge was the first symptom. In 4 cases there was slight transitory pain. The drumhead shows a grayish nodular infiltration in the early stages. This accounts for the common multiple perforations.

34. Ormerod, F. C.: J. Laryng. & Otol. **46**:449, 1931.

It is common to find two perforations, one at each side of the malleus. In 17 adult patients 8 had double perforations and one of these had double perforations of both ears. The uniformity in the site of the perforations is explained by the much poorer blood supply in the center of the drumhead. There is a tendency for the perforations to enlarge, coalesce and involve the whole of the membrane.

Granulation tissue is only seen in moderate amounts. The tubercle bacillus may be obtained from the ear with a special pipet. In 6 of 17 cases the bacillus was discovered in this way, and in one it was found by guinea-pig inoculation, though not by direct smears. Of these 6 patients 5 have since died of phthisis. All of the 11 patients in whom no bacilli were found were alive at the time of writing.

The mucous membrane of the middle ear is usually a pale pink. There is a tendency for the destructive processes to cease after a while. Mastoiditis is rare in adults.

Thomson [35] in the last four years found tuberculosis of the ears in less than 2 per cent of the phthisical patients in a sanatorium. It was slightly commoner in men than in women. He had studied 25 cases in twenty years. There was a striking absence of pain, fever or free discharge. The discharge was generally watery or curdy. There were only 2 cases of multiple perforations, and Thomson had never seen any complications. In the last 14 cases the discharge was examined for tubercle bacilli, which were found in 3 cases. In all of these there were bacilli in the sputum. In 3 of the 25 cases the perforation closed within a few months with restoration of the hearing. In the remainder the otorrhea persisted. It is a complication of evil omen. Most of the patients with otorrhea die within a few months.

AURAL INJURIES FROM FRACTURE OF THE SKULL

Davis [36] divided cases of aural injury due to fracture of the skull into three groups according to the degree of severity. The first group consists of hemorrhage into the middle ear only, without rupture of the drum. The membrane is bluish and there is a typical middle ear deafness. The lesion is probably a crack in the tegmen.

The second group shows a rupture of the membrane with bleeding from the meatus. The rupture is generally in Shrapnell's membrane. The roof of the middle ear may be comminuted, and spicules of bone may be seen in the meatus.

The third group consists of severe fractures involving the labyrinth capsule with escape of cerebrospinal fluid. These cases are usually fatal.

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35. Thomson, St. Clair: J. Laryng. & Otol. **46**:460, 1931.
36. Davis, E. D. D.: J. Laryng. & Otol. **46**:536, 1931.

Most of Davis' cases were of the middle ear type. If there is any improvement it usually occurs within eight weeks. Any deafness after that period is permanent. Davis had not seen any cases of genuine inner ear deafness resulting from fracture. Probably most of these patients die.

TUMORS

Cassidy [37] reported a case of tumor of the cerebellopontile angle on the left side. Functional tests showed spontaneous horizontal nystagmus, more marked to the left, and also some vertical nystagmus. There was a spontaneous pointing deviation to the left with the left arm, and there was falling to the left. When the head was turned to the right the patient fell forward and when it was turned to the left he fell backward. Rotation gave a lessened response and no pointing deviation when turned to the right, but when turned to the left the responses were normal. Stimulation of the right vertical canals [sic] gave the normal rotatory nystagmus, while stimulation of the left side produced a horizontal nystagmus to the right. This perverted inverse nystagmus indicated pressure on the brain stem. The caloric test of the right ear gave normal results for the horizontal canal but the vertical canals gave no response. There was almost no response on douching the left ear. The patient was subsequently operated on, with a good recovery.

37. Cassidy, W. A.: Ann. Otol., Rhin. & Laryng. **40**:178, 1931.

Abstracts from Current Literature

Ear

SEVERE INTOXICATION IN CLINICALLY LATENT OTITIS MEDIA. J. BONABA and A. NOGUES, Arch. de pediat. d. Uruguay **2**:341 (July) 1931.

The authors report a case in a girl aged 4 years. There were no obvious symptoms of otitis media. The illness began abruptly with severe vomiting, cough and irregular fever. The child showed great prostration, cyanosis, marked abdominal distention and enlargement of the liver. There were no symptoms in the lung. The Widal test and tuberculin reaction were negative, as were the results of examination of the spinal fluid. Spontaneous rupture of both ear drums revealed purulent otitis media as the cause. There was almost immediate cessation of all symptoms, with complete recovery. SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

EAR DRAIN. S. LAWNER, Monatschr. f. Ohrenh. **66**:7 (Jan.) 1932.

Lawner reviews the history of ear drainage, discusses the types of ear drain now in use and describes an ear drain invented by him, which he thinks meets all the qualifications for an effective drain. It consists of two parts, (1) a cone-shaped tube made of nonabsorbent cotton and (2) a bundle of fine, sterile, absorbent cotton wicks, which fills the bore of the tube and projects a few millimeters at each end. The actual drain, which consists of the wicks, has strong suction power and cannot become matted or lumpy, because it is stretched out in the tube. The fibers are soft and can be pushed up to the tympanic membrane by a gentle rotatory motion of the tube without irritation of the membrane. The tube itself remains dry because it is nonabsorbent, so that the wall of the auditory meatus is protected from contact infection. Its natural elasticity and rough surface prevent it from being displaced, and its conical shape prevents it from being pushed against the tympanic membrane in case of a forceful impact. Finally it can be introduced by the patient or by his relatives and thus permits the frequent changes necessary for the continuous drainage, which is important in the healing of otitis media and the prevention of complications. EDITOR'S ABSTRACT.

VESTIBULAR NYSTAGMUS AND CORTICAL INHIBITION IN PARALYSIS OF MUSCLES OF THE EYE. V. TANTURRI, Monatschr. f. Ohrenh. **66**:14 (Jan.) 1932.

Tanturri reports a case of paralysis of the ocular muscles in which vestibular nystagmus reactions were observed. Examination revealed (1) congenital paralysis of the lateral and medial rectus of the left eye, (2) spontaneous nystagmus of the right eye, (3) artificially produced horizontal nystagmus in both eyes in the medial position, (4) inhibition of the horizontal nystagmus in the left eye and continued nystagmus in the right eye when the patient moved the right eye laterally, (5) persistence of artificially produced rotatory nystagmus in the left eye in the medial position even when the right eye was making lateral movements. The most interesting features of the case were the vestibular nystagmus and the phenomena of inhibition. The author gives a comparative discussion of three similar cases reported by Baranyi and then discusses his own case, outlining the reasoning by which he arrived at a theoretical explanation of it. In this case, the author thinks the disturbance was in the posterior longitudinal bundle in the immediate proximity of the eye muscle nuclei (the left abducens nucleus from which the left lateral rectus is innervated and the right oculomotor nucleus from which the left medial rectus is innervated). Since the vestibular nystagmus is the result of a reciprocal, alternating rhythm of contraction of the active and relaxation of the antagonistic

muscle, and since voluntary lateral movements of the eye are also the product of muscular contractions and relaxation, one may assume that the summation and superimposition of such different stimuli can produce the nystagmic rhythm. If there is a disturbance of one or more tracts of the cortico-ocular and the vestibulo-ocular systems a series of complex phenomena of inhibition result.

EDITOR'S ABSTRACT.

SHOULD THE TERM TUBE OCCLUSION BE RETAINED? N. R. BLEGVAD, Monatschr. f. Ohrenh. **66**:32 (Jan.) 1932.

Blegvad criticizes the use of the term tube occlusion to designate a condition of the eustachian tube that results in the symptoms of baryecoia, pressure sensations, buzzing sounds and so on. This use of the term tube occlusion assumes an airtight closing of the lumen of the tube due to obstructions, such as adenoid vegetations, hypertrophic tonsils, scars and so on, and consequent thinning of the air in the tympanum, resulting in a retraction of the tympanic membrane, hyperemia of the mucous membrane of the labyrinth, formation of a transudate and the subjective symptoms already cited. On the basis of clinical experience, the author thinks this theory untenable, as its major premise, hermetic occlusion of the tube, could not be clinically demonstrated in any of his cases. The occlusion, which is always short of hermetic, is due to secretion in the tube and middle ear, and its degree depends on the amount and tenacity of this secretion. Proof of this is the fact that the occlusion sounds heard during air douche disappeared immediately after paracentesis of the tympanic membrane and the passage of air to the exterior could be heard. In a few cases in which the secretion was unusually tenacious, it required repeated douches to expel the secretion, but as soon as this was accomplished, the passage of air to the exterior could be heard. The symptoms that are supposedly due to negative pressure in the tympanum, resulting from occlusion, are due to other causes. The retraction of the manubrium is due to formation of adhesions between the manubrium and the promontorium and to possible shortening of the tensor tympani; the retraction of the pars tensa, since it continues after paracentesis, also cannot be considered an effect of negative pressure. The hyperemia of the wall of the labyrinth is due to a catarrhal condition of the mucous membrane. The fluid in the tympanum, although occasionally serous, is usually mucoid and sometimes tenacious and is to be considered an exudate, not a transudate. The frequent negative results of bacteriologic examination of the fluid may mean that the organisms are ultramicroscopic or hypovirulent. The author concludes that the condition designated by the term tube occlusion is an infectious process of the tube, originating in the nasopharynx and spreading to the tympanum, where it causes changes in the mucous membrane resulting in the symptoms of so-called occlusion. Since occlusion of the tube is not absolute, and since the involvement of the tube is only a part of the disease, the use of the term tube occlusion is incorrect. As the condition of the tympanum is analogous to that otherwise designated as catarrh, the author suggests the use of this term.

EDITOR'S ABSTRACT.

THE EAR IN TOWER HEAD. K. WITTMAACK, Monatschr. f. Ohrenh. **66**:44 (Jan.) 1932.

Wittmaack replies to Brunner's report on the anatomic examination in tower head in which the latter stated that the findings in the mastoid process could not be explained in terms of Wittmaack's pneumatization theory. To Brunner's statement that on the basis of the fibrous structure of the mucous membrane he expected, according to Wittmaack's theory, to find a diploid structure of the mastoid process, but found instead an almost sclerotic mastoid, Wittmaack replies that Brunner has misinterpreted the statement in his textbook. In this, he emphasizes that in a mastoid process of the structure described, which he thinks is due to an early cessation of the pneumatization process resulting from a fibrous

change of the superficial mucous membrane, one cannot expect such a pronounced diploid structure as in those cases in which the cessation of pneumatization due to fibrous change in the structure of the mucous membrane falls into a later period of development. In this book, he also says that this sclerotic structure is found only in a very small antrum, whereas Brunner considers the antrum in his case to be very large. However, Wittmaack thinks that the plates accompanying Brunner's report show that the antrum was in reality a small one. To the latter's statement that, in addition to fibrous and fibrous-hyperplastic mucous membrane, he found a region of normal mucous membrane, Wittmaack replies that this so-called normal mucous membrane resembled a fibrous mucous membrane more than a normal one. In cases in which it is difficult to judge the structure of the mucous membrane at one point, the morphologic development at this point should be considered as tending in the direction of that which is clearly manifest in the adjacent portions of the membrane, rather than in the opposite direction. Wittmaack thinks that the condition of pneumatization cannot be explained entirely on the basis of constitutional disposition, but is to be explained rather on the assumption that a certain phenotypic influence on the mucous membrane, of changing intensity and sometimes opposing form, took place during certain phases of development.

EDITOR'S ABSTRACT.

RELATIONS BETWEEN ROENTGENOLOGIC AND CLINICAL SYMPTOMS IN ACUTE OTITIS. K. EISINGER, Monatschr. f. Ohrenh. **66**:266 (March) 1932.

Eisinger discusses the relation between roentgenologic and clinical symptoms in acute otitis. Since mastoiditis usually occurs in a relatively well pneumatized mastoid process, whereas chronic otitis usually develops in cases with poor pneumatization, the roentgenologic visualization of the pneumatization type is of prognostic value in the interpretation of symptoms such as profuse secretions, pain, rise in temperature, etc., when these occur late in the course of the disease and the otitis has not shown any definite tendency to heal spontaneously. Absolute indications for operation, such as existing or impending intracranial complications, labyrinthine symptoms, paralysis of the facial nerve or periosteal abscess of the mastoid process, which demand intervention regardless of the stage of the otitis, supersede the indication of the roentgenogram. The disease of the sinus wall within the mastoid process is practically symptomless and can only be inferred from the clinical course, the condition of the middle ear and tympanum and the type and amount of secretion, but roentgenization sometimes makes it possible to find the explanation of these manifestations. However, the roentgenogram can never constitute an absolute indication for operation or be an indication in itself. In the early stage of otitis certain clinical symptoms have little influence on the prognosis and indication because they belong to the normal picture, whereas a roentgenologically visible infection of the bone in the early stage of otitis is of great prognostic significance. In the late stage every clinical symptom, though unimportant in itself, is of great significance, whereas a slight involvement of the bone occurring in this stage is not so significant. A positive roentgenologic observation opposed to an amelioration of clinical symptoms in case of protracted duration of the disease may be regarded as a lagging behind of the roentgenologic change (if the diagnosis of a mucosus otitis can be eliminated by clinical and bacteriologic examination); in mucous otitis, on the other hand, it is a valuable aid to therapy.

EDITOR'S ABSTRACT.

Pharynx

ARE THERE HARMFUL EFFECTS FOLLOWING TONSILLECTOMY? E. WIRTH and O. LACHENIT, München. med. Wchnschr. **78**:1898 (Nov. 6) 1931.

The authors reexamined ninety-two patients who had had their tonsils removed from one to ten years before, and did not find any harmful effects following the operation. Seventy per cent had no complaints. The others had minor complaints

due to conditions, usually in the nasopharynx or sinuses, that were present before the operation. The patients were asked if they would consent to a tonsillectomy, if they had the choice over again. Practically all answered in the affirmative.

BRAHDY, Mount Vernon, N. Y.

BLOOD PICTURE FOLLOWING SURGICAL INTERVENTIONS, ESPECIALLY FOLLOWING TONSILLECTOMIES. J. BOLAND, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:339 (Feb. 29) 1932.

Boland discusses briefly some of the literature on the effect of surgical intervention on the blood picture, and reports his own study of the blood picture following tonsillectomy in a hundred cases. The author's investigations were based on Schilling's hemogram table. Schilling differentiates between a degenerative and a regenerative nuclear shift. The regenerative shift is the one that Arneth calls the shift to the left. In agreement with the investigations in surgery and gynecology, the author always found a postoperative leukocytosis which disappeared in three or four days. The shift to the left was always of slight degree. In the onset of complication the change in the blood picture always preceded the change in the clinical picture by one or two days. The severity of the complication was parallel to the regenerative nuclear shift. The author also mentions that a lymphatic reaction of the blood was found before operation in most of the cases of chronic tonsillitis. He emphasizes the importance of serial examinations and the fact that a change in the blood picture is to be evaluated only as one of many symptoms constituting the entire clinical picture.

EDITOR'S ABSTRACT.

TECHNIC OF TONSILLECTOMY IN CHILDREN. HALLE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:380 (Feb. 29) 1932.

Halle states that he agrees with the general principles set forth by Ohnacker in his article on the technic of tonsillectomy in children. He thinks tonsillotomy may be employed only in cases in which there is a simple hypertrophy of the tonsil which constitutes a mechanical disturbance, and in that case one must be careful not to remove too much of the tonsil so that a merged tonsil, with its dangers of oral sepsis, results. He shares Ohnacker's views on indications for tonsillectomy, adding those cases in which there is fetid odor of the mouth which cannot be attributed to diseased teeth or gastric disturbances and especially those cases in which there is considerable swelling of the submandibular glands. In respect to the technic of operation, he finds the method outlined by Ohnacker a little more complicated than necessary and describes his own method by which tonsillectomy can be performed in a few seconds. It is based on Sluder's method, but makes use of the tonsillotome of Fahnenstock or Mathieu instead of the thick instrument of Sluder. To protect the second phalanx of the middle finger from the cutting action of the ring of the tonsillotome owing to the exertion of great pressure, he has devised a flexible ring that fits all fingers and that has a metal tongue over which the ring of the instrument is pushed. The steps of the operation are described; also how these must be modified in cases of adhesions of the tonsil. Halle advises keeping several long artery forceps on hand, but observes that they need rarely be used. Like Ohnacker, he performs adenotomy immediately after tonsillectomy whenever necessary. In most cases tonsillectomy can be performed without anesthesia; only when children are afraid does he employ a superficial ether anesthesia. He warns against an anesthesia deep enough to subdue reflexes, and says that if operation is undertaken with the head inclined slightly forward, swallowing of the adenoids or tonsil or an aspiration of blood or tumor particles will not occur. The author does not recommend the operation of Sluder for children over 12 years of age, as adhesions are more frequent then, and there is much greater danger of hemorrhage.

EDITOR'S ABSTRACT.

Larynx

INFECTIOUS LARYNGOTRACHEITIS OF CHICKENS. J. R. BEACH, J. Exper. Med. **54**:801 and 809, 1931.

The causative agent of infectious laryngotracheitis of chickens was found to be present in bacteriologically sterile tracheal exudate, spleens and livers of diseased fowls. The causative agent was present regularly in the tracheal exudate, in the spleens of about 60 per cent, and in the livers of about 30 per cent of chickens with active infectious laryngotracheitis. Suspensions of the spleen and liver were less effective in inducing the disease than those made from the tracheal exudate. This finding, with absence of pathologic changes in the spleens and livers, would seem to indicate that they are not actively involved, but that the causative agent is carried to them by way of the blood. The disease could, in our experience, he produced only in chickens. Domesticated ducks and several wild and free-flying species of birds, including sparrows, crows, starlings, doves and pigeons, were found to be refractory, and so, too, were rabbits, guinea-pigs, white rats and one pig that was tested.

Experiments have shown that tracheal exudate from two strains of laryngotracheitis of chickens from New Jersey and two from California when suspended in bouillon and passed through Berkefeld V filters will produce the disease. Two of six Berkefeld N filters allowed the etiologic agent to pass, whereas four did not. Attempts to produce the disease with Seitz filtrates were unsuccessful. These results demonstrate that laryngotracheitis is caused by a filtrable virus which because of its size or some other property does not pass readily through the finer filters. It has been shown that the serums from fowls that have recovered from an infection with one of the New Jersey viruses will neutralize the same strain and also the one California strain tested. In order to demonstrate neutralization conclusively it was necessary to titrate samples of dried virus and in the tests to use approximately ten infecting doses. The virus dried over calcium chloride for ten days and then stored in the refrigerator for sixty days produced disease. Kept over calcium chloride for a month it was still active and when dried by Swift's method it remained alive for five months. AUTHOR'S SUMMARIES.

HISTOPATHOLOGY OF INFECTIOUS LARYNGOTRACHEITIS IN CHICKENS. O. SEIFRIED, J. Exper. Med. **54**:817, 1931.

The characteristic lesions of infectious laryngotracheitis are ordinarily restricted to the respiratory tract and are most pronounced in the larynx and trachea. Sometimes the eyelids are affected. A certain percentage of the cases are associated with bronchitis and peribronchitis, and pneumonic areas and hemorrhages in the lung, while involvement of the nasal passages, communicating sinuses and eyes seems to be dependent on the mode of infection and the course of the disease. The virus affects the epithelial cells primarily, but soon inflammation develops in the submucosa and underlying parts. Edema is often extremely pronounced in the submucosa. The destruction taking place at later stages is due to edema, cellular infiltration and hemorrhages, and in some instances to secondarily invading bacteria. Characteristic intranuclear inclusions in the epithelial cells of the trachea are present in many cases. They bear a close resemblance to the inclusions occurring in herpes, varicella, virus III of rabbits and submaxillary gland disease of guinea-pigs.

AUTHOR'S SUMMARY.

CANCEROUS DEGENERATION OF PACHYDERMIA LARYNGIS. F. SPEISER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:391 (Feb. 29) 1932.

Speiser reports two cases of benign neoplasms of the larynx, one pachydermia and the other papillomatosis, in both of which after long years of observation the final change into a carcinoma could be followed. Both cases were combined with leukoplakia of the oral mucous membrane, indicating that pachydermia and

papillomatosis of adults belong to the group of leukoplakias of the mucous membrane. Speiser's own cases as well as a study of the literature show that differentiation of these two conditions is at times difficult, since clinical as well as histologic examination may give different results in the same case. The possibility of a cancerous degeneration is the same in both diseases. The change of these two conditions, clinically and histologically assuredly benign at the start, into cancerous neoplasms shows that both pachydermia and papillomatosis of the adult must be counted among the so-called precancerous conditions of the larynx. In view of the duration of disease in the two cases (from ten to twenty years) the author thinks that the pachydermia and the papillomatosis as such are not the sole cause of the cancerous degeneration, because these, as statistics show, are capable of spontaneous regression; rather, either these diseases created a local predisposition to cancerous degeneration, or a further cause such as age was added, inducing the change to malignancy. This confirms the new conception of precancerous growth, according to which the later is not a necessary phase of the cancer development but merely a local predisposition to such development. The frequent biopsies that were performed in these cases did not have any effect on the malignant degeneration, neither initiating nor exacerbating it, so there is no contraindication to this diagnostically and therapeutically important intervention to be inferred from these cases.

EDITOR'S ABSTRACT.

Nose

DIPLOPIA WITHOUT APPARENT LIMITATION OF OCULAR MOVEMENTS: RAPID RECOVERY AFTER THE REMOVAL OF A NASAL TUMOR. GENET and JACOD, Ann. d'ocul. **168**:669 (Aug.) 1931.

Genet and Jacod report a case in which diplopia developed in a patient in whom the only possible physical cause for the paresis of the left external rectus was a tumor in the nasopharynx. The diplopia and the paresis of the rectus disappeared the day following the removal of the tumor. The authors call attention to the fact that paresis of the oculomotor nerves is of longer duration when associated with infection in the nasal sinuses, and they believe that congestion was the probable cause of the paresis in their case.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.].

ROENTGENOLOGIC DIAGNOSIS OF MALIGNANT TUMORS OF NASAL SINUSES. E. G. MAYER, Monatschr. f. Ohrenh. **66**:303 (March) 1932.

Mayer thinks that while early roentgenologic diagnosis of malignant tumors is not as a rule possible, roentgenologic symptoms should be taken into consideration as there are cases in which these appear definite earlier than clinical tumors, also roentgenograms may throw light on the extension of the malignancy in cases in which clinical diagnosis is already definite. The changes a malignant tumor of the nasal sinuses effects in a roentgenogram may be divided into two groups, those having to do with the mucous membrane of the sinuses and those having to do with the pathologic changes of the bony skeleton. So far as the roentgenologic visualization of the conditions of the mucous membranes is concerned, the author thinks that a malignant tumor leads to an early complete shadowing of the diseased accessory sinus. The extension of the shadow in the pneumatic system of the nose does not give a basis for judging the extension of the tumor, as usually the sinuses adjoining the tumor are also shadowed due to a thickening of the mucous membrane or empyema. The existence of a partial shadowing in an accessory sinus is an indication that the tumor has not yet penetrated that sinus. Concerning the bony changes, the author says that, while roentgenologic manifestations of bone destruction are often uncharacteristic, they sometimes have a value in differential diagnosis. A well defined perforation of the wall of a sinus can be caused by an inflammatory process as well as a malignant tumor, but is more

often found in cases of malignant tumors. If a displacement of the bone or periostium at the edges of the defects can be demonstrated, this points with considerable certainty to an inflammatory process. An ill defined circumscription of the diseased sinus, as an expression of bone destruction, is found in inflammatory processes, as well as malignant tumors. If sequestration is demonstrable, it speaks with great certainty for osteomyelitis and against a tumor. If, in addition to the ill defined contouration of the diseased sinus, there is a diffuse osteoporosis, especially in the region of the nearby nasal skeleton, one may conclude the existence of a malignant tumor. Small, not sharply defined, yet rather well circumscribed foci of destruction in the region of the frontal sinus are characteristic of carcinoma. In estimating the extension of the tumor by roentgenologic symptoms of bony changes, one must take into consideration that defects of the walls of the sinuses rather frequently escape roentgenologic visualization, and a negative roentgenologic finding in a certain region is not proof that the tumor has not yet attacked that region.

EDITOR'S ABSTRACT.

RHINOLOGIC EXAMINATIONS OF THE SCHOOL CHILDREN OF BERN WITH SPECIAL CONSIDERATION OF OZENA. H. RAAFLAUB, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:359 (Feb. 29) 1932.

Raaflaub reports the results of rhinologic examinations conducted on 9,375 school children of Bern. The chief interest of the investigation centered in the occurrence of ozena. A diagnosis of ozena was made in all children with chronic inflammation and secretion of the nasal mucous membrane and pronounced atrophy of the inferior or middle turbinate though there was no fetor. Fetor is not a constant symptom of atrophic rhinitis; it is often missing, especially in children. Ozena occurs in 0.35 per cent of the school children of Bern. Up to the age of puberty it occurs with equal frequency in boys and girls, with the age of puberty it occurs about twice as often in girls. In 30 per cent of those affected, the secretion had a fetid odor. Ozena seems to occur more frequently in the poorer districts, also there are more children affected among backward pupils than among those of normal ability. The Wassermann reaction was negative in the twenty-one cases in which a test was made. Enlarged pharyngeal tonsils were found in 37.4 per cent of the normal children and 48 per cent of the backward children. In the fifth year of school the pharyngeal tonsils were enlarged in 47.3 per cent of all the children examined; in the ninth year, in 27.5 per cent. The palatine tonsils were enlarged in 41 per cent of the children in the fifth school year, and in only 31 per cent in the ninth school year.

EDITOR'S ABSTRACT.

METHOD OF RADICAL OPERATION OF MAXILLARY SINUS WITH REFERENCE TO POSSIBLE INJURY TO TEETH AND OTHER COMPLICATIONS. HALLE, Ztschr. f. Hals, Nasen- u. Ohrenh. **30**:386 (Feb. 29) 1932.

Halle disagrees with the statement made by Nagel in his article on injuries to the teeth following radical operation on the maxillary sinus, that the injuries to teeth incurred by Denker's method are too slight to warrant substitution of a new method for this time-tested one. The author thinks that injuries to the teeth are not rare in this operation or even in the Canfield-Sturmann operation through the nose, which the author considers less destructive than Denker's method and equally successful. The author has had cases in which paresthesia of the teeth lasted twenty years and was considered more unpleasant by the patient than suppuration; in isolated instances patients complained of severe neuralgic pain. Halle thinks that the surgeon should choose that method which is the most conservative though as successful as others. He discusses again his own method of radical operation on the maxillary sinus, which he considers less destructive than the methods of Denker and even of Canfield-Sturmann and least likely to be attended by injuries to the teeth. The approach is through the nose. An excellent view of all but the anterior wall of the sinus is obtained and this can be easily con-

trolled with a Ritter curet or a curved spoon. If desired, it may be seen by use of a little mirror, or one can remove just enough bone from the anterior edge of the crista piriformis so that the front wall can be seen. The necessity of exercising care in removal of the diseased mucous membrane in the lower anterior angle of the sinus has already been stressed by Nagel. The healthy mucous membrane of the nose is preserved in its entirety. Of the bone, only the part adjacent to the nose below the inferior turbinate is removed, but it contains no important nerves or vessels. In the temporary chiseling off of the lateral nasal wall immediately behind the crista piriformis, nothing is removed or destroyed and the wall heals back in place soon after the operation. The operation can be executed with a minimal reaction and without visible swelling of the soft parts. The healing process in all cases has been complete in one or, at the most, three weeks following the operation. The technic is not difficult for the skilled rhinologist.

EDITOR'S ABSTRACT.

Miscellaneous

TREATMENT OF MALIGNANT NEOPLASMS OF THE ESOPHAGUS BY MEANS OF RADIUM. F. J. CLEMINSON, Monatschr. f. Ohrenh. **65**:1406 (Nov.) 1931.

Cleminson discusses seventy-five cases of malignant neoplasms of the esophagus, including ten tumors in the hypopharynx or in the postcricoidal region. For the treatment of the latter, he employed an operative method of implantation of radium needles posterior and exterior to the pharynx. Further, he describes various methods of application of radium and of radon and also different kinds of containers for these, which were employed in the treatment of tumors of the esophagus proper. The duration of life, after the first examination at the clinic, was, in the patients with postcricoidal tumors, 7.3 months, and in those with esophageal tumors, 6 months. The patients with esophageal tumors recently have shown a prolongation of life. The author thinks that it is better to give several irradiations than to give only one.

EDITOR'S ABSTRACT.

INFERIOR VOCAL ORGANS. J. NÉMAI, Monatschr. f. Ohrenh. **65**:1451 (Dec.) 1931.

Némai believes that chronic hyperemia and inflammation of the larynx are not solely due to external irritants such as smoke, alcohol, frequent colds, etc., but that anatomic variations in the larynx may lay the foundation for chronic inflammation and inferiority of the vocal organs. If there is any anatomic defect, no matter how slight, which presents an obstacle to the perfect closing of the rima glottidis, the muscles have to put forth an extra effort to overcome this hindrance and hyperemia, fatigue and deficient function of the vocal organ results. The author describes four types of anatomic variations of the larynx which he considers predisposing factors for inferior vocal organs. The first is premature ossification of the cartilagenous structure of the larynx. If the thyroid notch is deep, it compensates for loss of elasticity of the cartilage, but if it is shallow, the rigidity of the cartilage becomes apparent early and voice production is restricted. The second variation described is asymmetry of the larynx resulting from an asymmetric fusion of the laminae of the thyroid cartilage. The rima glottidis is distorted, and usually the vocal folds do not meet at the same elevation. The third variation described is the ventral elevation of the vocal folds so that they form an acute angle with the ventral wall of the larynx instead of the customary right angle. The air pressure at this point is increased, and it requires an extra effort for the muscle to keep the rima glottidis closed. The fourth anatomic variation is the incomplete closing of the rima glottidis leaving a small funnel-shaped aperture at the dorsal end of the opening. This aperture which the author has called "fovea subglottica" is concealed from above by the arytenoid columns, but may occasionally be seen through the tracheal fistula in patients on whom tracheotomy has been performed and has been demonstrated by the author in necropsy

specimens. In this type of anatomic variation, adduction may be sufficient for voice production, but an extra burden is placed on the muscles, resulting in hyperemia and finally pachydermis laryngis. EDITOR'S ABSTRACT.

INDICATIONS FOR ROENTGEN THERAPY IN RHINOLARYNGOLOGY. R. GOLDMANN, Monatschr. f. Ohrenh. **66**:52 (Jan.) 1932.

Goldmann recommends the use of roentgen therapy to rhinolaryngologists because he finds that in many cases it combines effective treatment with the least possible distress and inconvenience to the patient. Foremost among diseases in which roentgen therapy is indicated are acute inflammatory conditions. The danger imputed to the destruction of the protective lymphocytic wall is contraindicated by clinical experience. In this respect, dosage is important, and any dose that locally destroys the cells necessary for reparation, osteoblasts, granulocytes and so on, is to be avoided. As the effect of roentgen therapy on the general condition of the patient is an important aid to its local effect, successful therapy demands that the infectious process shall not have advanced so far that the patient has lost the capacity to react. Among the acute inflammatory conditions in which Goldmann has successfully used roentgen therapy are: furuncles at the entrance to the nose, facial erysipelas, uncomplicated acute tonsillitis, phlegmon of the peritonsillar region and the floor of the oral cavity, also the rigidity of the jaw that sometimes follows these conditions, and rheumatic myositis. Roentgen therapy has also proved successful in acute inflammation of the nasal sinuses. The method to be used in irradiation of a sinus is described. In cases of chronic inflammation, roentgen therapy meets with some difficulties, owing to the persistence of the focus of infection, as well as to local and constitutional predisposition, and must therefore be accompanied by treatment aimed at the primary cause; thus, in cases of hypertrophy of the tonsils, irradiation of the tonsil must be preceded or accompanied by treatment intended to combat the disposition to tonsillitis. In cases with tonsillar plugs, roentgen treatment causes a decrease in the formation of plugs and in the inflammatory conditions, especially the lymphadenitis, also in the focal infections caused by the plugs. Increased resistance of the tonsillar and surrounding tissue to bacteria and their toxins seems to play an important part in this process. The author thinks roentgen treatment of diseased tonsils is indicated wherever there is no urgent need for tonsillectomy and in cases in which there is a contraindication for operation. In acute cervical lymphadenitis, roentgen therapy is useful if accompanied by causal therapy and inclusion of all the cervical glands in the field of irradiation. In chronic cases, a possible portal of entry is to be destroyed prior to instituting therapy. In tuberculosis of the larynx, only the hypertrophic, nonulcerating type may receive roentgen treatment and that only in very small doses. EDITOR'S ABSTRACT.

HISTOLOGIC REGRESSION OF EPITHELIOMAS OF THE LYMPH GLANDS FOLLOWING ROENTGEN THERAPY. A. TOBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:182 (Dec. 18) 1931.

Tobeck contributes a discussion of the question as to whether epitheliomas of the lymph glands may justly be classified in a separate group, or whether they are to be considered as carcinomas with secondary lymphocytic infiltration. He gives a negative answer based on the histologic effect of roentgen treatment of a tumor of the palatine tonsil diagnosed as an epithelioma of the lymph glands. Three treatments were given, and biopsies were made before roentgenization, between treatments and at intervals for one month thereafter. Roentgenization was followed macroscopically by a rapid disappearance of the tumor. Microscopically, it was followed by a rapid disintegration of the epithelial components of the tumor accompanied by a relatively good preservation of the lymphocytic components. The rapidly developing granulation tissue was at first rich in plasma cells and later, during cicatrization, in mast cells. In view of the histologic effect

of roentgenization on the rapid disintegration of the epithelial components, preservation of the lymphocytic components and the rich development of cells removable by lymphocytes, the author considers the separate classification of epitheliomas of the lymph glands of doubtful justification. He thinks that epitheliomas of the lymph glands are rather to be considered as carcinomas composed of young, slightly differentiated and therefore roentgen-sensitive epithelial cells, with secondary infiltration of lymphocytes.

EDITOR'S ABSTRACT.

EFFECT OF TRACHEOBRONCHOSCOPY ON BLOOD PRESSURE AND ON RESPIRATION. T. J. ABRAMOW, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:197 (Dec. 18) 1931.

In reviewing the literature on bronchoscopy, the author finds that much has been written on the technic of this method but very little about its immediate effect on the cardiovascular system and the respiration of the patient. The author reports the results of tracheobronchoscopies conducted by him on fifteen dogs. Tracings of the blood pressure and the respiratory movements made during the bronchoscopies showed a decided drop and fluctuation in the blood pressure and a change in the rhythm and depth of respiration. Severing of both superior laryngeal nerves and both vagus nerves resulted in remission of the respiratory and cardiovascular reaction to bronchoscopy. This indicates that the observed reactions are reflexes produced by irritation of the branches of the superior laryngeal and vagus nerves in the mucous membrane of the respiratory tract. The author found that shortening the duration of bronchoscopy to a minimum does not prevent the appearance of serious disturbances in blood pressure and respiration. Cocaine anesthesia of the mucous membrane of the respiratory tract, in temporarily removing the reflexes on the heart and the respiratory organs, greatly simplifies the execution of tracheobronchoscopy and is to be recommended for all cases.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Feb. 1, 1932

HOWARD C. BALLENGER, M.D., *President, in the Chair*

LEUKEMIC CHANGES OF THE UPPER RESPIRATORY TRACT AND EAR. DR. RICHARD H. JAFFÉ (by invitation).

When I was asked to address the Society and speak of my experiences at the Cook County Hospital, I went over the records and was struck by the frequency of changes in the upper respiratory tract that I found in cases of leukemia. Certain observations, I think, are particularly interesting.

The leukemic changes in the upper respiratory tract can be divided into two groups. I should call the first the inflammatory ulcerative group and the second the tumor-like group. The inflammatory and ulcerative changes in the upper respiratory tract in leukemia occur in association with similar lesions in the oral cavity or independently of such lesions. The latter holds true especially if the teeth are absent. The changes usually start in the tonsils, spreading to the pillars of the tonsils, to the back of the tongue, to the pharynx and epiglottis, and even to the larynx and trachea.

There is a thick greenish-white membrane covering the tongue, soft palate, pillars and tonsils, a gangrenous, sloughing, foul smelling, dirty, grayish exudate which comes off easily or irregular deep ulceration.

The first stage of these changes is a hemorrhagic extravasation, as part of the generalized hemorrhagic diathesis. These hemorrhagic areas become infected, gangrenous and ulcerated because in leukemic conditions the defense reactions of the body are diminished. Thus, hemorrhages change into a pseudomembranous and gangrenous inflammation and, after sloughing of the dead tissue, into ulcers.

The inflammatory, ulcerative lesions occur most frequently in the acute form of leukemia. It is a familiar fact that one may distinguish between acute leukemia and chronic leukemia. Some investigators believe that acute and chronic leukemia are different conditions, but I believe that they belong in the same group. Ulcerative and gangrenous changes are less frequently found in the chronic form, but if they are found they are usually the first sign of a shifting from the chronic stage into the acute stage of the leukemia. When, in a case of chronic leukemia, ulcerative or gangrenous lesions suddenly appear on the tonsils or pharynx, one can be sure that the acute stage is imminent.

The second type of change in the upper respiratory tract in leukemia is the tumor-like infiltration of the tissues by the leukemic cells. Most of these cases show the first changes in the tonsils. Usually the tonsil on one side starts swelling, and often this swelling later extends down to the pharynx and to the epiglottis. I have seen leukemic infiltration in the larynx and in the trachea with severe respiratory complications. The infiltrations are quite firm, and on microscopic examination are seen to be composed of leukemic cells. In a few cases the leukemic changes occur in the uvula before other manifestations of the leukemia are visible.

Most of the cases of leukemic infiltration of the upper respiratory tract that I have seen were cases of chronic lymphatic leukemia, in particular the so-called aleukemic form.

It is not correct to speak of the condition as leukemia, which means "white" blood." Leukemia is not a disease of the blood itself but of the blood-forming organs. Therefore, one should speak of lymphadenosis if it is a lymphatic leukemia, or of myelosis if it is a myeloid leukemia. Then one can differentiate between the leukemic and the aleukemic forms. I have found that the most extensive proliferations occur in the cases of aleukemia. In the examination of the blood one finds a slight anemia, say, 4,900,000 or 3,500,000 red cells, with a hemoglobin content of 60 or 55 per cent and from 8,000 to 10,000 white cells, and the differential count is practically normal. In this form of leukemia a careful examination of the blood is required to recognize the leukemic condition. I think that in the routine examination of the blood these cases are often missed. If one finds from 30 to 40 per cent lymphocytes, not much significance will be attached to this differential count, for many persons have a relative lymphocytosis throughout life. In an aleukemic lymphadenosis, however, the finding of 30 or 40 per cent lymphocytes is not normal. The majority of the lymphatic cells are larger than normal lymphocytes, the media staining lighter and being surrounded by a more abundant cytoplasm.

The chronic forms of leukemia usually occur between the ages of 40 and 70. In some of the cases in which I performed an autopsy the first symptom was an enlargement of the tonsils. In one case the tonsils were removed and the patient bled to death. This is of great importance. The danger of severe hemorrhage after tonsillectomy in cases of leukemia is tremendous, and the difficulty of recognizing the aleukemic condition is great. How can the diagnosis be secured? Perhaps it can be made by means of a biopsy, but even so it is not always possible to make the diagnosis of leukemia, since the differentiation from simple hyperplasia may be exceedingly difficult. Enlargement of the lymph nodes is of the greatest differential diagnostic importance, though the enlargement may be only slight. I have seen aleukemic infiltrations in the pharynx in which the lymphadenopathy was hardly noticeable. An unaccounted-for enlargement of the tonsils in an elderly person is always suggestive of leukemia.

The next question is the treatment of the leukemic infiltrations by radium and roentgen rays, and here again there are great dangers. When one treats an aleukemic lymphadenosis with x-rays, one is apt to change the aleukemic lymphadenosis into an acute lymphatic leukemia. Suddenly, two or three days after the first roentgen treatment, the white count jumps to 70,000 or 80,000, a hemorrhagic diathesis develops and the patient dies within a few weeks.

Involvement of the tonsils is occasionally found in a condition that is related to leukemia but differs from typical leukemia by the distinctly malignant character of the infiltrations. I mean the so-called "leukosarcomatosis." This name was introduced by Carl Sternberg. What does lymphosarcomatosis mean? It is really a lymphosarcoma with a leukemic blood picture.

The other condition in which I have found the most extensive infiltrations of the upper respiratory tract is chloroma, which is characterized by the greenish color of the newly formed tissue. The local process is quite destructive, much more so than in lymphadenosis or myelosis, and the blood picture is either that of a lymphoblastic or a myeloblastic leukemia.

I have never seen leukemic infiltrations of the mucosa of the nose. I think that they are extremely rare, although they have been reported. Inflammatory lesions are much more common. Not long ago, I studied a case of subacute lymphatic leukemia in a child, aged 6 years, whose first symptom was a persistent rhinitis with a hemorrhagic discharge. The mucous membranes of the nose were covered with a thick membrane and foul smelling crusts, which resisted every treatment. A diagnosis of diphtheria was made, and antitoxin was injected. An acute hemorrhagic diathesis developed, and the child died within a few days.

On looking over the records I found two more cases of lymphatic leukemia in children which started as acute rhinitis. Otitis media is very common in leukemia. In 10 per cent of all cases of leukemia in which we were permitted to open the skull we found an otitis media characterized by extensive destruction of the bone.

In one of my cases of leukemia the first symptom was sudden deafness in one ear. The difficulty in hearing developed several weeks prior to an enlargement of the abdomen and the other symptoms of leukemia. Paralysis of cerebral nerves is not rare in leukemia, and it affects especially the acoustic and facial nerves. In these cases one finds either hemorrhages into the nerves or leukemic infiltrations, the latter particularly in chloroma. In the cases of chloroma the infiltrations develop near the bone, and may extend along the acoustic nerve into the ear. This is a direct extension, just as in cases of meningitis in which the infection spreads into the internal ear.

According to Wittmaack, so-called hemorrhagic fibrinous labyrinthitis is found most frequently in leukemia. It is not so much a labyrinthitis as a hemorrhage into the perilymphatic spaces which causes a collapse of the endolymphatic spaces, the hematoma later becoming organized by granulation tissue, which in turn may be followed by leukemic infiltrations. In other cases the granulation tissue may become ossified.

Other changes that occasionally occur in leukemia are hydrops of the endolymphatic spaces of the labyrinth, with extreme distention, and leukemic infiltration in the ganglion vestibulare.

DISCUSSION

DR. T. C. GALLOWAY: It is fortunate that we have such presentations as this by Dr. Jaffe to keep alive our contact with other branches of medicine. We like to be lazy and think that if we know conditions in the ear, nose and throat that is all we need, and that the internists and pathologists will keep us out of trouble. I think this discussion shows that we must always be alive to the fact that no condition can be diagnosed as purely local until systemic causes have been ruled out. This applies especially to necrotic lesions, quinsy, ulcerative stomatitis, scurvy and agranulocytic angina. In particular, no diagnosis of Vincent's angina is sufficient. In practically all these ulcerative lesions one will find Spirilla. If one is content to stop with a diagnosis of Vincent's angina, one is likely to get into trouble. We have all had experience with such cases as Dr. Jaffe mentioned, in which an apparently innocent condition proves to be much more. Recently a general practitioner called me to see a supposed quinsy sore throat. There seemed to be a good deal of edema and the patient looked very ill; before incision, therefore, a blood count was ordered, and a chronic leukemia was found. Another atypical tonsillitis proved to be an acute lymphatic leukemia. Recently at the Cook County Hospital Dr. Robert Lennon and I had a case that was unique in my experience. A cursory search of the literature does not give an exact duplicate. A man came with a probable diagnosis of carcinoma of the esophagus, the usual history of difficulty in swallowing, loss of weight and strength, and x-ray findings of an obstruction about 8 cm. below the suprasternal notch. Endoscopy showed an infiltrated, red, granular stenosis which looked not unlike a malignant growth. Only a no. 12 Jackson bougie could be passed. A biopsy was performed and, owing to a clerical error, the report was given to me as carcinoma, though the pathologist had called it chronic inflammation. Assuming the growth to be carcinoma, we prepared for a diathermy operation by placing packs along the esophagus through incisions in the neck. A gland external to the lesion removed at this time was diagnosed by Dr. Jaffe as showing leukemia, as was a subsequent biopsy. On the basis of the blood picture he called the case aleukemic leukemia. A radium capsule with 50 mg. of the element was placed above the lesion for ten hours, after which the patient's obstruction diminished rapidly and he insisted on going home. He died at home three weeks after leaving, without any subsequent check-up or autopsy.

I think it is well to recall to you that at the Cook County Hospital on Thursday morning Dr. Jaffe conducts a clinicopathologic conference, which is one of the most instructive things in medicine in this country. All physicians are welcome, and a wealth of clinical and pathologic material is most interestingly and instructively presented.

DR. SAMUEL SALINGER: When we were preparing the questions for the Cook County examination for attending men in the nose, ear and throat department, we were trying to think up some very difficult questions. It occurred to me that we might ask, "What are the temporal bone changes due to leukemia?" The question was not used because the examining men knew so little about it, but I still think that it would have been a good question.

Some years ago I had a case that impressed me with the value of always having in mind the unusual. A woman had a peculiar velvety membrane which extended over both tonsils and onto the pillars. This membrane was easily stripped off, but recurred within a few hours. She was toxic, and had so much difficulty in breathing that we performed a tracheotomy, but we were still at sea on the diagnosis until I happened to talk to Dr. Vaughan about it. He said: "That is easy. It is one of the forms of acute leukemia and we get one here about once a year." The patient died shortly after the condition was verified by examination of the blood.

DR. ALFRED LEWY: I should like to have Dr. Jaffe tell us the differentiation between aleukemic leukemia and agranulocytosis, on the one hand, and what was formerly called the infective mononucleosis on the other. I have difficulty in making the clinical differentiation. I should like to know whether the presence or absence of a hemorrhagic tendency can be used as a differential point.

DR. E. W. HAGENS: Some years ago the American Otological Association was presented with a collection of slides on pathologic changes of the ear. It has been housed at Rush Medical College. In that series there is a leukemic slide, and as I remember the leukemic cells infiltrated the perilymphatic spaces and also the endolymphatic. I do not remember that there was pressure on the ductus cochlearis, but will look it up. We have that series of specimens there, and they are available to all members of the Society. I think that Dr. Shambaugh would be glad to have any one come out to see the slides who is interested.

DR. RICHARD H. JAFFÉ: In regard to the case that Dr. Galloway mentioned, with the mediastinal lymphadenopathies compressing the esophagus, these cases are interesting because the condition resembles carcinoma so much, especially if the enlarged glands cause decubitus ulcers in the esophagus.

As Dr. Salinger mentioned, the cases of leukemia are not very rare. During the last three years 3,000 necropsies were performed at the Cook County Hospital, among which there were 28 cases of leukemia.

Agranulocytosis is not a disease entity, but a complex of symptoms. This complex may occur under different pathologic conditions, most frequently, however, as I believe, in septicemia. It is doubtful whether there exists an "essential or primary agranulocytosis." Some cases of agranulocytosis followed injections of arsphenamine. Millions of injections of arsphenamine are given every year, but agranulocytosis develops in only a few instances. It is difficult to explain this fact. It seems to me that both agranulocytosis and leukemia are abnormal reactions of the bone marrow to a severe, usually infectious irritation. In agranulocytosis the reaction is anergic, while in leukemia it is hyperergic. Back of these abnormal reactions is the peculiar constitution of the bone marrow.

Hemorrhagic diathesis does not occur in typical cases of agranulocytosis. It is always suggestive of acute leukemia or aplastic anemia; in the latter instance the bone marrow is depleted of all blood-forming elements. Infectious mononucleosis is a benign condition characterized by the complete absence of changes of the red blood cells and a high percentage of irritation forms, derived from the lymphocytes.

CASE PRESENTATIONS: UNIVERSITY OF ILLINOIS, SCHOOL OF MEDICINE

Cordopexy in Bilateral Abductor Paralysis of the Vocal Cords. Dr. Noah Fox.

Mr. M. M., aged 24, gave the following history: In October, 1930, he was treated by his family physician for a sore throat which was not severe enough to keep him in bed. On the fifth day of the illness, he began having respiratory difficulty, and was sent to the City Contagious Hospital for intubation. This proved unsatisfactory, and a tracheotomy was done. Numerous smears taken from the larynx at this institution failed to reveal the Klebs-Loeffler bacillus until the third day of his stay there, when a positive smear was obtained, but no membrane was ever present.

He remained there several months, during which time attempts at decannulation were unsuccessful. Finally, in January, 1931, the tube was removed and the patient sent home. His breathing remained labored and stertorous, especially at night or when he walked a short distance, making it impossible for him to work.

When he was examined in September, 1931, a bilateral abductor paralysis of the cords was noticed, the latter lying in the cadaveric position.

A tracheotomy was done, and several days later a cordopexy after the method of Irwin Moore. It was thought that perhaps anastomosis of the transplanted thyro-arytenoid with a group of muscles enervated by the hypoglossus might keep up the tone of the thyro-arytenoid muscle, so the latter was attached to the bellies of the thyrohyoid and sternohyoid muscles, and anchored by a guide suture to the outer integument of the neck. The tracheotomy tube was removed after three weeks. At present, four months after the operation, the patient is quite comfortable. He is able to exercise normally and carry on his daily work. He has had several colds, and although his voice has been affected each time, he has not had the slightest respiratory difficulty.

Bilateral Abductor Paralysis. Dr. Noah Fox.

Case 1.—T. L., a woman, aged 34, was admitted to the hospital in March, 1931, complaining of a choking sensation in the throat, noise in the throat following exertion, insomnia, nervousness and palpitation. A thyroidectomy had been performed seventeen years before.

Examination showed a bilateral abductor paralysis. An emergency tracheotomy was done a week later, without much improvement. Then a laryngofissure with a hemicordectomy was done, followed by excellent results.

Case 2.—M. S., a housewife, aged 52, entered the clinic in February, 1931, complaining of marked shortness of breath. She had had a thyroidectomy seven years before.

Examination showed a bilateral abductor paralysis of the vocal cords. A tracheotomy was first done and then a laryngofissure, with the removal of the right true and false cords. The postoperative course was good.

Case 3.—E. A., a woman, aged 52, was first seen Aug. 3, 1931, complaining of hoarseness and great difficulty in breathing. A thyroidectomy had been done on March 22, 1931, and the hoarseness occurred immediately following operation. The wound became infected and had to be reopened, and prolonged drainage ensued. The hoarseness and difficulty in breathing had increased to the point of great embarrassment.

Examination showed a bilateral abductor paralysis. An emergency tracheotomy was performed under adverse conditions; namely, the patient was short-necked, the depth of tissue between the trachea and skin was great and much scar tissue was present as the result of the infection. A tracheotomy tube of unusual length had to be employed.

PARALYSIS OF THE RECURRENT LARYNGEAL NERVE. DR. NOAH FOX.

The frequency with which paralysis of the recurrent laryngeal nerve follows in the wake of operations on the thyroid makes this complication one of major importance in thyroid surgery.

The types encountered are: 1. Paresis of one or both cords, which is not permanent; it may be due to stretching and slight surgical trauma, pressure of dressings or hematoma and infection. It clears up in weeks or perhaps years. It also occurs from stretching of the nerve during surgical anesthesia.

2. Unilateral involvement. The cord on the affected side will remain fixed in an intermediary position. The voice will be comparatively good because the other vocal cord compensates, and when one cord functions respiration is not interfered with. When no operation has been performed, these cases usually are due to carcinoma of the thyroid gland, benign enlargement or intrathoracic goiters.

3. Bilateral involvement. A serious clinical problem occurs when both recurrent laryngeal nerves are concerned in the process. Here the patient is in grave danger of asphyxiation, especially if an acute infection ever involves the larynx. This is an abductor paralysis and causes the cords to become fixed in the midline, which is the phonatory position. It is for this reason that the voice is not too greatly affected in this type.

It is of importance to mention that according to deQuervain, the recurrent laryngeal nerves occur anterior to the inferior thyroid arteries just as frequently on both sides, and they penetrate the thyroid space a little farther from the tracheo-esophageal groove than is usually described. Therefore, to avoid injury to these nerves, extrafascial ligation of the inferior thyroid artery is more reasonable.

The reason given why adduction instead of abduction occurs following section of the inferior laryngeal nerves is that it has been histologically proved that the greatest degree of atrophy occurs in the abductor fibers.

Treatment consists of (1) emergency tracheotomy, (2) permanent tracheotomy, (3) cordectomy, (4) cordopexy or (5) nerve anastomosis.

CEREBROSPINAL RHINORRHEA. DR. NOAH FOX.

Miss B. L., aged 33, gave the following history: In the early part of March, 1930, she contracted what appeared to be an ordinary head cold of possibly a little more severity than she had ever had. Especially prominent were the symptoms of sneezing and nasal discharge. No headache had ever been present. The cold lasted for about two weeks, but she noted that after all other symptoms had subsided, a clear fluid continued to drip from her left nostril, particularly when she bent her head forward. The nasal drip had been present about four weeks when she was first examined. Nasal examination gave entirely negative results, all changes from the previous cold having disappeared. However, a clear watery fluid was found to be dripping from the nose, so that it easily filled a 20 cc. test tube in about thirty minutes. The fluid was clear, and had a specific gravity of 1.008. On the addition of acetic acid no precipitate appeared. Boiling failed to produce a coagulum, but the addition of magnesium sulphate to saturation caused precipitation. The addition of nitric acid caused a precipitate to form, which disappeared when the solution was boiled. With a trace of copper sulphate and an excess of potassium hydroxide the solution turned pink. When it was boiled with Fehling's solution, a reduction occurred.

In order to ascertain further the character of the fluid, 5 cc. of 0.6 per cent indigo carmine was injected into the spinal canal. A bluish-green coloration of the nasal drip appeared after fifteen minutes, and continued for about five hours. Nasal examination at this time revealed the colored material dripping over the lateral wall of the left nasal cavity in the region of the carina and agger. We assumed that the separation in continuity of the cribriform plate had occurred anteriorly where it might be easily accessible to intranasal treatment.

In seeking an etiologic factor, several stereoscopic plates both in the lateral and anteroposterior positions were taken, but appeared negative. The eyegrounds and visual fields appeared normal. The obesity of the patient suggested hypophyseal disease, but no evidence of this has yet occurred. At the suggestion of Dr. Joseph Beck an attempt was made to set up a fibrosis in the area involved, which might result in occlusion of the separation. The roof of the nose was swabbed every two weeks with a 20 per cent solution of silver nitrate. After six applications fibrous bands began to appear, and the discharge diminished until it entirely stopped, five months after treatment was begun. It has remained dry for five months. The patient is apparently normal and without either nasal or intracranial symptoms.

MALIGNANT GROWTHS ABOUT THE HEAD AND NECK. DR. FRANCIS L. LEDERER.

Apart from the usual problems involved in the treatment of cancer, malignant growths about the head and neck offer special difficulties due to their exposed location and the involvement of sense organs. The cosmetic results and loss of sense of sight and of speech are of necessity major problems.

The site of the tumor may prevent early recognition, but too frequently, owing to such factors as a lack of radical intervention at the onset, these growths may so progress as to be placed in an inoperable group. We look askance on a case in which roentgenotherapy has been employed, not because of any thought that roentgenotherapy is not beneficial when properly indicated, but because too much time is allowed to elapse and the disease is permitted to progress.

In our opinion the factors that make a tumor inoperable are: first, the location of the tumor; second, metastatic glands, and third, surgical timidity. The first two conditions have been alarmingly difficult because involvement of the hypopharynx, esophagus, base of the tongue, bronchiogenic regions, etc., has taken a steady toll despite our efforts. In regard to the third point, we can only show a few successes, but feel that we have been amply repaid for our efforts.

The rehabilitation of these patients is an important item too often ignored. Surgically this is a most interesting and fascinating phase, and should not be delegated to the oral or general surgeon. We have been interested in other methods of restoration, such as protheses of various types. Special indications exist for this form, and various technical problems are involved that cannot be considered at this time.

CASE 1: *Sarcoma of the Antrum.*—J. K., a man, a candymaker, in 1928, at the age of 39, had a small sore at the right side of his nose, which lasted about six months. The lesion became larger and bled. He was operated on at another hospital, and also had some radium treatment. Six months after the onset of the trouble, there was a large encrusted mass presenting from the right nostril. The right cheek was infiltrated, and the skin was under tension. The mouth showed involvement of the hard palate on the right side. A biopsy was performed, and showed fibrosarcoma.

Under synergistic analgesia the common carotid artery was ligated on the right side. Using the Mouré external incision along the side of the nose, under the orbit and through the lip, the entire superior maxilla was removed, including the infiltrated and involved skin over the cheek. The skin of the upper lip was allowed to remain. The patient was fed by nasal catheter for about a week, and made an uneventful recovery. About a year and a half later an Italian skin graft was taken from the right biceps area and the flap attached to the face. The patient was kept in this position by a plaster cast for about two weeks. The skin was then severed from the arm and was attached to the margin of the deficient area on the face, no epithelial lining being used in the buccal cavity. Since this time, a number of steps have been taken to get more complete union of the flap. The patient has gained 37 pounds (16.8 Kg.) since he was first seen.

CASE 2: *Carcinoma of the Orbit.*—J. H., a carpenter, in 1926, at the age of 71, had an epithelioma of the orbit involving the upper and lower lids at the

inner canthus on the right side. He had been treated for a number of years with radium plaques. In 1926 the growth was removed surgically, and later electrocoagulation was used. In 1928, there was a recurrence, the pathologists reporting a squamous cell carcinoma with early hornification. At this time the involvement was so great that sacrifice of the eye became imminent. The patient did not return to this service until some months later, when he reported that he had been to an institute in which some arsenical paste was used; in a short time this had destroyed quite a large area, including the entire eyeball and the eyelids. He reported that he was not very uncomfortable with this treatment, except for a short period of time. When he returned, there were defects leading into the lateral wall of the nose and into the frontal sinus. In November, 1931, a flap was raised from the forehead in the preliminary procedure; about two weeks later the pedicle was swung over the orbit and nose, areas that had been excised, and the deficiencies were closed over.

CASE 3: *Carcinoma of the Nose.*—J. B., a man, aged 61, a musician, presented himself at the clinic in the summer of 1930, giving a history of repeated attacks of swelling of the nose for one year, and of a destructive process on the inside of the nose for the past four years. He was seen by a number of physicians in Chicago, and, despite a negative Wassermann reaction, was thought to have a chronic granuloma of a syphilitic nature. Repeated studies of tissue did not reveal any specific process. A biopsy was performed, which showed a reticulated type of structure, thought to be an undifferentiated squamous cell carcinoma. Later, sections taken from areas that had not been subjected to the various medications showed positively a squamous cell carcinoma.

The external nose was widely removed, including a great part of the septum, which had been involved in the destructive process. A surgical procedure, together with surgical diathermy, was used. Avertin anesthesia was employed. Healing was ideal, and in May, 1931, a full thickness graft was swung down from the forehead and attached to two lateral flaps, swung in from the cheeks. This pedicle healed nicely by first intention, and was then severed from the forehead. There has been another procedure since that time, and a graft to build up the tip is contemplated in the near future.

CASE 4: *Carcinoma of the Mandible.*—C. W., a deputy sheriff, aged 51, was first seen in the department of oral surgery in 1927. In March, 1931, he came in with a swelling of the jaw of three years' duration. He complained of pain like a dull toothache. A slight nodule on the right side of the lower jaw, which had been treated by electrocautery three years before, was now elevated to the size of a walnut and was of a fungating character. This mass was seen on the inside of the mouth, and involved the buccal mucosa of the cheek.

A pathologic diagnosis of squamous cell carcinoma of the right side of the lower jaw was made, and a resection, combined with surgical diathermy, was performed. The postoperative course was good, and the patient was discharged for deep roentgenotherapy. The mouth has remained clean, the patient has good function and he has gained in weight.

CASE 5: *Carcinoma of the Lip with Intensive Operative and Radiation Destruction.*—In B. E., a man, aged 37, a clerk, a sore developed on the lower lip eighteen months before examination. He thought the lesion merely a cold sore, but it did not respond to the usual conservative therapy. Six months later a diagnosis of cancer was made, and a small V-shaped resection of the lip was performed. There was primary healing, but a month later there was evidence of local recurrence. The patient then went to the Mayo Clinic where the whole lower lip and the glands of the neck were removed. This was followed by intensive roentgenotherapy and radium therapy. Following the irradiation, the patient was quite ill and had to remain in bed for three weeks. Extensive destruction and sloughing of the tissues of the chin and submental area resulted from the irradiation. In the early part of December, 1931, the patient came to the Research Hospital, showing an exposed mandible which appeared devitalized. The teeth that still remained were loose and rather painful. There were areas around the jaw that

had characteristics of granulation tissue. Biopsy specimens were taken from eight or nine different areas surrounding this terrain, all of which were negative for carcinoma. Roentgenograms of the mandible confirmed the clinical opinion that the bone was not viable.

The patient was built up and studied for some time. In the middle of January, 1932, it was decided to begin to obtain material for closing up the defect. A flap was taken from the left side of the chest, down to the fifth rib, and was brought up and attached to a hairy flap taken from the scalp on the left side of the head. These were sutured together in the manner of a tube, according to the method of Gilles, the idea being to line the mouth with the smooth skin from the chest, and to have the external layer made up of the hairy portion of the scalp in the manner of a beard. The possibility of preserving the lower jaw is still a moot question.

CASE 6: *Audible Speech Following Laryngectomy.*—J. T. B., a sales manager, aged 63, early in the month of April, 1924, noticed occasional hoarseness which caused no pain or discomfort, but which persisted from day to day. He was otherwise in good health. He consulted numerous physicians, whose opinions varied, and a number of months passed. He had a grayish mass on the left vocal cord. Radium treatment was applied externally, with no apparent benefit. He was then hospitalized, and radium was applied directly to the larynx, with no added improvement.

In June, 1924, the patient became alarmed at the increasing hoarseness. On consulting numerous specialists, he was advised by one to have a hemilaryngectomy, by another to have radium therapy and by others to use watchful expectancy. In the middle of July, 1924, he consulted Dr. Robert C. Lynch, who confirmed the diagnosis of squamous cell carcinoma by biopsy and then performed a laryngectomy. He was able to return home and resume his duties within four weeks.

At this time he first consulted Dr. Lederer for after-care. Despite instruction in the technic of producing sound, audible speech was not possible for more than four years following the operation. A fairly good whispering voice or the use of pad and pencil sufficed. During this time the patient tried to create sound by belching or by forcing air from the esophagus. By diligent practice he was able to learn this form of speech so that it was audible, fluent and even possessed modulation. He was able to use the telephone, acted in the capacity of salesman and readily dictated correspondence.

Analysis of the patient's speech showed that he produced consonants by the muscles of the mouth and cheek, bringing air in the mouth under certain pressure, in contact with the articulating narrowing. He had a vicarious or pseudoglottis in the esophagus which was stretched and narrowed at will, being impinged on by swallowed or inspired air. From five to ten syllables were spoken, and then came the swallowing act.

CASE 7: *Retropharyngeal Abscess; Periesophageal Abscess; Mediastinitis, Followed by Esophageal Stenosis.*—Mrs. W. L. K., aged 29, a physician's wife, was seen on Nov. 11, 1930, complaining of pain on swallowing and a fetid discharge from the pharynx. She gave the following history: Three months before she presented herself (Sept. 7, 1930), pain on swallowing developed; this seemed to have come on about twenty-four hours after she had eaten a club sandwich. There was a sensation of pressure at the second intercostal space. For four days she experienced this discomfort in swallowing. On the fifth day she was seen by Dr. Zinn of Baltimore, who examined her fluoroscopically by x-rays and by esophagoscopy. His fluoroscopic examination showed an obstruction to the bismuth capsule opposite the third and fourth dorsal vertebrae. Esophagoscopy revealed a swollen mucosa of the posterior esophageal wall at this point, bulging forward. No foreign body was found. The patient's condition seemed temporarily better. That night there was a sudden onset of pain in the back and chest and inability to swallow water. The neck became swollen, the temperature rose to 103 F., and the blood count showed 25,000 leukocytes. The patient was given morphine hypodermically, nutritive enemas and dextrose intravenously but nothing by mouth.

Twenty-four hours later the swelling was marked and she was having considerable trouble in breathing, evidently due to pressure. The lungs were normal except for the left upper portion which showed some dulness extending to the midsternal line. The swelling and difficulty in breathing were becoming more marked, with evidence of laryngeal involvement. The patient was unable to open the mouth. The substernal dulness on the left side was more marked.

The blood picture showed a steady rise in the polymorphonuclear cells, leukocytes and white cells. As the patient's condition was becoming progressively worse, Dr. Zinn decided to attempt evacuation of the abscess which he felt was present. Two attempts at esophagoscopy had been made. Roentgenograms of the spine and chest were negative.

On September 15, under avertin anesthesia, Dr. Zinn made an incision into a large abscess in the upper mediastinal region, removing from 3 to 4 ounces (85 to 113 cc.) of foul smelling pus. The patient immediately showed improvement, with a drop in the number of white cells from 26,000 to 12,000 and in the polymorphonuclears from 93 per cent to 76 per cent within a few days. The abscess drained profusely for fourteen days, and then the patient was able to swallow small amounts. Dr. Zinn reported: "Several doctors were in consultation and they were also puzzled as to the exact cause of the abscess. I have had one other such case, which proved fatal, and on autopsy was unable to find any definite cause except a large abscess posterior to the esophageal in the upper mediastinal."

When the patient came under my observation, swallowing was still difficult and painful. She had some limitation of movement of the head, being unable to flex, extend or rotate the head freely; she swallowed more easily when the head was flexed on the chest. The patient was aware of a fistula in the pharynx about a month and a half after the original opening of the abscess. This opening was seen to the left of the midline on the posterior pharyngeal wall, opposite the epiglottis. It was surrounded by a smooth, rounded, nipple-like projection, about one-fourth inch (0.6 cm.) in diameter. Creamy yellow pus was seen to come from its crater. There were no other findings in the pharynx or larynx. There was an apparent swelling of the neck, not unlike that of a thyroid enlargement. On study of this fistulous opening with radiopaque substances, it was seen to extend retroesophageally to the region that had previously been described by Dr. Zinn. Despite the fact that the roentgenogram did not disclose a foreign body, I was guided by the previous opinion that a radiotranslucent foreign body was still present. Consultants recommended an external approach into the prevertebral space to drain the abscess; however, as a conservative procedure was urged, the fistulous tract was curetted in the early part of December. The patient did not have a severe attack until two weeks later, when pus evidently accumulated again and then was discharge spontaneously. These attacks continued with decreasing severity and at longer intervals until April, 1931, when the process evidently subsided. Since that time the patient has been having increasing difficulty in swallowing so that now only the finest morsels and fluids can be taken, owing to a longitudinal stricture with a circular band of scar tissue at the upper end of the esophagus.

As an aftermath of all this, Dr. Zinn was asked to furnish the previous history and the roentgenograms that were taken when the patient was seen by him. The lateral view showed a definite retropharyngeal abscess, overlooked, no doubt, owing to the history of the possible ingestion of a foreign body and to the extreme condition present at the time of first examination.

Carcinoma of the Tympanomastoid Process. Dr. John Theobald.

W. W., a man, aged 49, a factory worker, presented himself in 1929, complaining of dull pain in the left ear of about fourteen months' duration. He had been under the care of one of the members of this department for a suppuration of fourteen months' duration. There were some resistant granulations coming from the middle ear, which did not respond to cauterization. A section taken at that

time showed a squamous cell carcinoma, a diagnosis which I confirmed. The mass was at first confined to the annular ring where there was a perforation. Later, while the patient was under observation in the clinic, the mass extended into the auditory meatus up to the region of the isthmus. A radical procedure was recommended, and the patient was told that the external ear might be sacrificed. Under avertin anesthesia, the usual procedure on the mastoid was carried out; the mastoid cells themselves showed little disease, but the middle ear was filled with the fungoid mass. A radical mastoid procedure was carried out, and by means of surgical diathermy the entire membranous canal was removed and the cavity thoroughly coagulated. For a number of months the patient made an uneventful recovery; then he began to complain of some indefinite gastric symptoms. He was referred to the department of surgery, where a diagnosis of possible gastric carcinoma was made, and a complete gastrectomy was performed. The involvement proved to be an adenocarcinoma. The patient made a rapid and uneventful recovery. About a year later, some of the exposed bone of the mastoid that had been fulgurated and had a tendency not to heal was removed, and a plastic operation was performed to close up the exposed retro-auricular area. The patient had been very comfortable since that time, and had shown no evidence of recurrence.

AMYLOID TUMORS OF THE UPPER AIR PASSAGES. DR. P. A. HALPER.

This pathologic condition is rare; it occurs as a part of a generalized amyloidosis, in which other organs are involved, or as a local condition. Amyloid is a homogeneous protein substance deposited in the tissues the origin and nature of which are obscure. It can be definitely differentiated by its appearance, color reactions and the condition under which it occurs. The albuminous material may be present in the tissues in such small amounts as to be recognizable only under the microscope, or it may be so abundant as to give a very characteristic appearance to the tissue. It most frequently occurs in the smaller arteries and capillaries the lumina of which are encroached on by the thickening of the walls that the process involves. It is usually the media and intermediary layers of the intima that are earliest and most extensively affected. The change also occurs often in the interstitial connective tissue and membrana propria of organs and in reticular connective tissue. It does not affect the parenchymal cells of organs. These, however, undergo pressure atrophy. The upper air passages more frequently than any other part of the body are involved in a local amyloidosis. The amyloid tumors are divided into three types: (1) diffuse subepithelial infiltration, (2) tumor-forming local amyloidosis and (3) amyloid degeneration of a preexisting tumor.

Burrow and Neumann, in 1873, were the first to recognize amyloid tumors of the larynx. These were found at autopsy. In 1875, Ziegler at autopsy found multiple amyloid tumors of the tongue and larynx and believed them to be of syphilitic origin. In a review of the literature up to 1919, New was able to collect forty-two cases of amyloid tumors of the upper air passages, and added four more, which he discussed in detail in the *Laryngoscope* (**29**:327 [June] 1919), making a total of forty-six cases. Since 1919 the literature regarding this condition has been rather scant. The four cases of amyloid disease were found among 217 neoplasms examined at the Mayo Clinic. In the forty-six cases collected, the ages ranged from 20 to 80; about half of the patients were between 51 and 70. In all the cases reported up to New's article, males were affected three times as often as females. All New's patients were women.

Symptoms of amyloid tumors of the upper respiratory tract vary with the location of the tumor. In many of the cases reported, there were no local symptoms, but the condition was accidentally found at autopsy. When the larynx and trachea are involved, the symptoms are such as those which accompany benign neoplasms of slow growth.

Amyloid tumors of the upper air passages are most common in the larynx. They are found in the larynx three times as often as in the trachea, which comes next in order of frequency. In one case reported, the larynx, trachea and bronchi

were affected. Tumors had occurred in the nose, the tongue and the tonsils. Local amyloidosis of the upper air passages occurs most often in nodule form, as in the case here reported. Multiple or single nodules, pedunculated or sessile, develop in the tumor, forming local amyloidosis. Amyloid degeneration may develop in a preexisting tumor, such as a fibroid, or in an angioma.

The diagnosis must, as a rule, be made microscopically, although general amyloidosis occurs following certain chronic disorders, such as syphilis, tuberculosis or osteomyelitis. The cases collected are of local amyloidosis without a degenerative process, but the condition may be due to chronic inflammation elsewhere in the body. In one of New's cases, there was an associated pathologic condition, a chronic cholecystitis for which an operation was performed. In the case presented here there is a history of syphilis, contracted two and a half years before, for which the patient was treated. His serologic tests are now negative.

In many cases the tumor appeared as a small nodule on a vocal cord and was readily removed perorally. In cases of a localized tumor, such as ours, surgical removal by endoscopy, indirect laryngoscopy or thyrotomy is indicated. When the disease involves the entire circumference of the larynx and trachea, the removal will probably result in loss of the voice and necessitate the permanent use of a tracheotomy tube because of secondary contractions. There was one case reported of entire disappearance of the tumor after two x-ray treatments. Dr. New had employed fulguration and radium in three cases with good results. In the fourth case, a thyrotomy was done because the diffuse infiltration involved the circumference of the larynx and trachea.

Amyloid Tumor of the Larynx.—S. B., a waiter, aged 42, was admitted to the hospital in October, 1931, complaining of hoarseness of many years' duration. The patient gave a history of syphilis contracted two and a half years before, which was treated. The Wassermann reaction is now negative.

Examination showed thickened cords and a reddened granuloma on the right true vocal cord, and a pedunculated tumor coming from the anterior commissure. Under local anesthesia a fibroma-like tumor was removed by direct laryngoscopy. The pathologic study was most interesting: "An ovoid nodule, 3½—2 mm., composed chiefly of coarse trabeculae of a homogeneous, hyalinic material. This material stains rose red with congo red and purple with violet methyl violet. Between the trabeculae there is a scanty amount of loose fibrillary connective tissue with single fibrocytes. The nodule is traversed by an endothelial lined sinusoid blood space."

RADIOSENSITIVE CARCINOMA OF THE ANTRUM. DR. M. R. GUTTMAN.

J. McK., a housewife, aged 39, was seen in November, 1931, with a definite swelling over the right side of the face extending over the jaw and involving the right lower lid and conjunctiva. Biopsy of tissue taken from the right antrum revealed the transitional cell type of carcinoma. The bone was eroded, and an infiltration of a whitish-gray tissue into the right cheek was noted. The patient was advised to have deep roentgenotherapy.

Radiosensitive Tumor of the Ethmoid Sinuses.—W. H., a man, aged 30, a cook, entered the hospital in June, 1931, complaining of nasal obstruction which had been present on the right side for one month and on the left side for eighteen months. Bulging of the left eye was first noted one month previously, and there had been a serous, bloody discharge from the left side of the nose for three months. Polyps had been removed from the nose during the preceding year, with a diagnosis of large spindle cell sarcoma for which radium therapy was given. There had been no loss of weight.

In the right side of the nose examination revealed polyps of a mucoid type high up in the middle meatus. The left side was completely obstructed by a polyp of fibrous type and a bloody seromucoid discharge. There was evident proptosis of the left eye.

Biopsy at this time showed what was believed to be a large round cell sarcoma. Because clinical and x-ray studies showed bilateral involvement, a course of deep roentgenotherapy was given. The reduction of the swelling with the diminishing of the tissue in the nose was remarkable, and further tissue study disclosed a transitional cell type carcinoma. The patient remained relatively free from discomfort for some months, but was now showing a tendency toward more rapid growth of the tumor.

DISCUSSION

DR. T. C. GALLOWAY: The question has been raised about the varying radiosensitivity of tumors and the course to pursue with these growths. I think that Dr. Jaffe could give us some valuable information, and I would like to hear his classification of tumors in relation to the use of radium, roentgen rays diathermy and other means of treatment.

DR. RICHARD H. JAFFÉ: To classify tumors according to their radiosensitivity is difficult. It is known that certain tumors respond promptly to radium or roentgen ray treatment. I mention only lympho-epithelial and transitional cell carcinoma of the pharynx and nasopharynx and Ewing's tumor of the bones. But histologic structure and radiosensitivity do not always go together. Two histologically identical tumors in two different persons may react quite differently to radiating energy. This indicates the great importance of the host, of the entire organism, for the curative effect of radium or roentgen ray, a factor so often neglected in modern literature. I also do not think it advisable to divide the tumors into separate groups according to their malignancy, using the microscopic structure as an index, and to speak, for instance, of a squamous cell carcinoma, grade II, etc. How often we find the greatest variations in the structure of a malignant tumor! One field may be classified as grade I, and another field, a few millimeters away, may be classified as grade IV.

DR. SAMUEL SALINGER: I think that we should all be grateful to the University of Illinois for presenting this wonderful group of material. In spite of the variety, I still do not know what to do for progressive deafness. I am glad that Dr. Fox was successful with silver nitrate in his case of rhinorrhea for it gives me a remedy that I hope to use on some of my cases of cracked brains that worry me to death.

I have read of many different methods of treating abductor paralysis. The last that I read was a report about transplantation of the omohyoid. I think that it was fastened to the muscle facet of the opposite arytenoid, the idea being that it could be made to serve as an abductor. That was done only experimentally. I think that the case reported by Dr. Fox is the first that I have ever heard of in a human being. I should like to know whether he used the technic to which I have referred or whether the muscle was connected directly to the vocal cord.

DR. T. C. GALLOWAY: I think that we should express our gratitude to these men for showing us this splendid group of cases, for it makes us see that such patients are not beyond hope. They presented a group of very bad cases with excellent results. I think that it speaks well for the future of otolaryngology in this section that such work is being done.

IRRIGATION OF THE ANTRUM BY WAY OF THE INTERNAL OPENING. DR. WALTER H. THEOBALD.

Motion pictures were presented demonstrating the technic and the advantages of this method as compared with the use of the trocar which necessitates puncture.

THE ACUTE COMMON COLD. DR. CHARLES B. YOUNGER.

This disease, which has grown from comparative insignificance to a pathologic problem of the first magnitude, increases in seriousness and importance when we consider its far-reaching possibilities. It probably constitutes the greatest single

cause of partial disability or incapacitation. It has been referred to by King as "the billion dollar disease." The common cold is a factor that enters into the work of every physician, and may complicate any illness. It should never be disregarded or treated lightly. Our conviction is that the acute common cold is an acute systemic disease, the cause of which varies. It is not primarily or specifically a disease of the nose and throat, and it is not due to a simple type of infection. Much work has been done on the subject of the etiology, which remains unsettled.

Because of lack of definite information as to the etiology, little advancement has been made in the methods of treatment. Certainly a good physique must be a safeguard, no matter on what hypothesis the acute cold may be explained. An abundance of sunlight applied to as much of the body as possible is recommended. In the winter months the quartz or mercury vapor lamps may be employed. Regular bathing and regular exercise are approved. Clean habits, clean, wholesome, well lighted living quarters and sleeping conditions suited to the individual must receive attention. Sleeping rooms that are warmed, but have proper circulation of fresh air, are generally favorable for sensitive skins and mucous membranes. Sleeping in drafts should be avoided. Clothing suited to the mode of life of the individual is important in prophylaxis. A study of a hundred patients treated by vaccines at the Massachusetts General Hospital showed good results in 68 per cent, the stock vaccines being just as effectual as the autogenous.

The condition of the nose and throat sometimes plays an important part in the matter of prophylaxis, a disturbance of their function giving rise to congestion and eventually to inflammation. Deformities, congenital or acquired, septal deviations, hypertrophied turbinates and diseased sinuses, tonsils and adenoids may be a positive hindrance to a proper functioning of the nose and throat membranes. In such cases correction is indicated. The subject of irritants and allergy in relation to colds will not be discussed in this paper.

At the onset of an acute cold, efforts should be directed toward restoring as quickly as possible the normal functioning of the body processes. Rest and relaxation are indicated, with frequent rest in bed. Extremes of heat and cold should be avoided. For the promotion of diaphoresis and diuresis, hot drinks are advised within moderation, and a mild laxative may be indicated. Purging is contraindicated. Headache, muscular pain and other symptoms may suggest a proper drug or other therapy. For the nasal symptoms palliative treatment is the best that we have to offer. In the early, congestive stage of the coryza it is not my habit to use or prescribe aqueous sprays or douches. The patient may be given some relief by the judicious use of epinephrine or ephedrine in the form of either an ointment or an oil solution. After the congestive stage has passed, alkaline solutions may be used to free the nasal passages from the thick, tenacious mucus. At this stage and later, mild silver preparations and other medicaments may be of benefit. Generally speaking, efforts in the acute cold are best directed toward correction of the systemic disturbances.

DISCUSSION

DR. HORACE LYON: Dr. Younger has outlined a disease or a symptom complex that, in its final analysis, is most peculiar. The cold is by far the most common of all afflictions, yet it remains essentially a mystery. Four decades ago, with the advent of improved microscopes in the hands of an increasing number of students and scientists, it seemed reasonable to assume that the cause of colds would soon be found. These four decades, however, have not revealed the etiology of colds; in fact, many investigators feel that the signs and symptoms are an expression of some other change, and that therefore a cold is not a disease. There is much evidence supporting a chemical etiology of colds. The normal chemical balance in the blood and body tissues is disturbed, and cellular metabolism is altered. Experimentally, under rigid control, the symptom complex of colds has been produced when it was reasonable to assume that new germs were not introduced as an etiologic factor.

Supporting the infective theory of colds is a mass of evidence within the experience of everyone. It is known that the disease is contagious, and one assumes that by contact a person acquires a new germ or a different type of some well known organism and so contracts a cold. Also one knows that a cold is frequently produced by severe chilling of the body without the introduction of any organism.

The various measures that help to prevent colds, for example, proper clothing, food, ventilation, etc., are known. These measures are not directed to the cold in particular, but are general measures to maintain or increase one's natural resistance to infections. Vaccine therapy has been a disappointment. Some persons receive striking benefits, but the percentage of improvement is small.

The direct local treatment for acute colds has also been disappointing. Certainly shrinkage and drainage to the nose and local medicinal applications and heat for the pharynx are beneficial but not specific. Rest in bed seems of greatest value.

The complications of colds in mortality and morbidity are appalling; in fact, these complications constitute the great bulk of the illness prevailing during the winter months. It is generally recognized that the most outstanding need at present is the discovery of the etiology of the common cold. With this accomplished, it is reasonable to assume that a more specific form of treatment will be developed which would prevent many of the serious complications.

DR. AUSTIN A. HAYDEN: I have enjoyed Dr. Younger's paper very much. The subject does not permit of any more general statements than he has made. One generality that he suggested was that we should go into the homes and change the sleeping conditions. That covers a lot of territory.

One thing he left out, although I think it is extremely important to the laryngologist. He said nothing about blowing the nose during an acute cold. I believe this to be very important, as it leads to many involvements of the sinuses, middle ear, etc.

DR. ROBERT SONNENSCHEIN: There was a statement in Dr. Younger's paper that even among the Eskimoes colds are present. I recall that in the late Nicholas Senn's book he stated that the Eskimoes did not suffer from ordinary "head colds" until white men went up there and gave them the infection. The reason I mention this is that the laity have the idea that cold weather produces head colds. I always tell them it is not the cold weather but the foolish mode of dressing. In England colds are not nearly so prevalent for the reason that the houses are not overheated as ours are. We bundle up; children and adults sit in warm rooms or go into heated stores and then go directly out into the cold. When patients or doctors say that they think it better to wait and not operate on the head or throat in cold weather, I tell them that I see no difference between results in winter and summer, unless there is prevalence of an epidemic of acute infections of the upper respiratory tract.

As to the treatment, I think that we shall have to agree that the vast majority of cases are self-limited. The old German statement that if one treats a cold carefully it gets well in a week, and if one does not treat it it gets well in seven days is still true in most cases.

DR. CHARLES B. YOUNGER: I did not mean to have Dr. Hayden take me too literally in suggesting that we go into other people's homes and regulate their living arrangements, but it is well within the scope of our work to see that conditions within the homes of our patients are conducive to successful treatment. In many cases when children are in poor health as the result of repeated or continuous colds, one may find on investigation that they are living, and particularly sleeping, under conditions that are decidedly to their disadvantage. This applies to cases in adults as well, with special reference to ventilation and exposure to cold air and drafts during the night.

Dr. Hayden also called attention to the dangers of blowing the nose. No doubt this is improperly done, and too vigorously in many instances, and should

be corrected. This can well be a part of a child's training. Complications that arise in connection with coryza may in many cases be properly attributed to this very thing.

In the matter of climate, as referred to by Dr. Sonnenschein, I believe that persons develop a constitutional resistance or tolerance to their environment, according to where they live, as in the arctic regions, England, or elsewhere. That is where the heat-regulating apparatus of the body comes into play.

March 7, 1932

HOWARD C. BALLENGER, M.D., *President, in the Chair*

SOME UNUSUAL NEURO-OTOLOGIC CASES. DR. SHERMAN SHAPIRO.

Oculogyric Crises.—Dr. Shapiro examined three cases of sudden spasmodic ocular deviations in patients referred with a diagnosis of chronic postencephalitic parkinsonism. In all of them the cochlear function was found to be normal. Vestibular tests made between attacks revealed no spontaneous vertigo, nystagmus, past pointing or falling. Responses to labyrinthine stimulation gave normal horizontal and horizontal rotary nystagmus in all; in one case rotation with the head over the shoulder induced normal downward nystagmus but no upward nystagmus. Perverted vertigo sensation, abnormal or diminished past pointing and falling and diminished susceptibility to stimulation were found in all. Attacks were witnessed in two cases; in one case the deviation was upward and to the left and was accompanied by a dissociated nystagmus; in another the deviation was horizontal. The deviation could not be reversed or could be reversed only with difficulty by irrigating the homolateral ear. In discussing these cases, Dr. Shapiro expressed the opinion that these phenomena were essentially of a vestibular character but above the level of the brain stem. He believes that the vestibular findings are in accord with the experimental work of Muskens, who has demonstrated connections between the terminal nuclei of the posterior longitudinal bundle and the pallidum and has called attention to the similarity of these forced ocular movements to the forced movements in animals following injuries of the vestibular tracts.

Hyperesthesia Acoustica.—This concerned a man, aged 40, who suffered from pain in both ears on exposure to noise. When the noise was very loud and he was exposed for a considerable time, the pain went to the top of his head and he suffered from typical vertigo. The symptoms first came on during battle maneuvers in 1917. Inspection of the ears showed retraction and degenerative changes in both drum membranes. There was a bilateral hearing impairment of the inner ear. Vestibular examination gave negative results except for some evidence of hypersensitiveness. Dr. Shapiro made tests with both the Kobrak minimal caloric test and the galvanic test both before and after exposure to noise and found that nystagmus responses were definitely increased following noise. The case is interesting from the fact that noise produces not only trigeminal but also vestibular symptoms. A study of the physiologic and anatomic factors involved indicates that the mechanism of this conversion cannot be lower than the optic thalamus and probably takes place in the cerebral cortex.

Cerebral Syphilis Complicated by Trauma.—This patient, a man, aged 51, had sustained a severe injury to the head two years previously with prolonged unconsciousness. Since his accident he had suffered from severe vertigo, especially on change of position. Neurologic examination disclosed absent patellar and Achilles' tendon reflexes, but no other findings. The Wassermann reaction of the blood was positive; that of the spinal fluid was negative, but the fluid gave a positive Pandy reaction and an increased cell count. There was a moderate

bilateral deafness of the inner ear. Vestibular examination disclosed the following: an occasional sustained first degree horizontal nystagmus to the right with a spontaneous drift (Abweichs-Reaktion) reaction of the outstretched arms in the same direction; a vertical nystagmus downward of coarse amplitude, which was accompanied by marked vertigo and could be elicited only when the patient was lying on his right side; there was also a spontaneous past pointing in the horizontal plane; i. e., the patient, when bringing his arm in from the side, persistently pointed under the examiner's finger. The caloric and galvanic tests showed a tendency for much more marked responses to occur when nystagmus was induced to the right. When cold water and the anodal current were used, the left side gave the strongest responses; when hot water and the cathodal current were used, the right side predominated. This is a nystagmus tendency in the sense of Kobrak and Vogel and is characteristic of a central involvement. The other responses to caloric and rotation tests were normal. The picture presented by this man is that of a vestibular disharmony in two planes, horizontal and sagittal, and the lesion is placed in the higher portion of the brain stem on the right side. The use of Frenzel's illuminated glasses as a help in studying spontaneous nystagmus was advocated.

DISCUSSION

DR. SIGMUND KRUMHOLZ (by invitation): The subject of oculogyric crises, to which Dr. Shapiro has directed attention, is, to my mind, most timely and important. This symptom occurs quite often as one of the sequels of epidemic encephalitis. It is characterized by transient conjugate deviation in any direction of the eyes alone or, which is more frequent, together with the head. There are a number of theories concerning the mechanism of this phenomenon, and the study of its reflex arc is now most opportune and, if established, would be a most valuable aid in the localization of various cerebral lesions. In contradistinction to the conjugate deviations that occur in acute diseases of the brain, the oculogyric crises are by far more accessible to investigation. They recur quite frequently; they are of sufficient durability, and the patients are otherwise fairly healthy and can be submitted without harm to various tests. If we are to take advantage of the oculogyric crises in the study of their reflex mechanism, it is most important to do so now while the symptom is still prevalent. Epidemic encephalitis, like noma, will probably in another generation pass into the domain of medical history, and the oculogyric crisis—one of its sequels—will then be no more at our disposal for investigation.

Dr. Shapiro has described the extensive experimental studies of Muskens, which demonstrate the dependence of the various conjugate deviations on the vestibular tracts and centers as well as on their fibrous connections with the globus pallidus. In his diagrams he has pointed out the probable pathways in the posterior longitudinal bundle as well as in the supravestibular centers, which constitute the reflex arc of these forced ocular movements. It is generally recognized that the posterior longitudinal bundle with its connections is the tract of the reflex mechanism for the associated ocular movements and that it most probably brings the vestibular apparatus into relation with the globus pallidus.

Prevost in his time pointed out that the conjugate deviations of the eyes that occur in lesions of the frontal region and in the angular gyri of the parietal convolutions are due to the involvement of the basal ganglions. Recently attention has been called to the fact that ocular conjugate deviations are indicative of an ipsolateral cerebellar lesion, and contralateral conjugate deviations are diagnostic of cerebral disease. In abscesses of the brain, in which you gentlemen are vitally interested, this phenomenon could be a most valuable aid in localization of the suppurative process.

The vast amount of clinical material that epidemic encephalitis has offered for the study of the extrapyramidal system has helped to a considerable extent to clarify our understanding about many of the clinical patterns produced by lesions of the basal ganglions. The oculogyric crisis is a sequel of epidemic encephalitis

that occurs quite frequently. I have now under observation six or seven cases, some at the Cook County Hospital and others at the Northwestern University Medical School. Undoubtedly there are a large number of cases at the Oak Forest Institution. Therefore, the present time to study the reflex mechanism of this phenomenon is most opportune. I wish to commend Dr. Shapiro for the interest he is taking in this symptom from the neuro-otologic angle, and may I urge you gentlemen of the neuro-otologic specialty to utilize the oculogyric crisis in the search for its reflex arc?

Dr. Alfred Lewy: We have been treated to a demonstration of advanced or higher otoneurology, higher at least in that it involves a higher portion of the brain than otologists are accustomed to investigate. I think Dr. Shapiro deserves great credit for his study of the anatomic work, but you noticed he confined his comments to Muskens' investigations, which are recent and not entirely corroborated by other investigators. Lorento de Nó found that section of the longitudinal bundle did not interfere with nystagmus by stimulation of the labyrinth. He did find that section of the arcuate fibers did so, and his general conclusions, based on a long series of experiments, were that many of the vestibular phenomena are not carried by any particular strand of fibers, but are really worked over in the substantia reticulosa and are not due to certain tracts. These findings are not altogether in agreement with those of Spiegel, so I do not know, and I do not suppose any one will know until further investigations are made, just how these impulses are conducted. As Dr. Krumholz pointed out, the phenomena of oculogyric crises are analogous to, or perhaps the same as, conjugate deviation, which is a phenomenon with which we are a little more familiar clinically. Conjugate deviation, as Dr. Krumholz pointed out, occurs in lesions of the posterior fossa, in lesions of the temporal lobes and in lesions elsewhere, and I think that it also occurs in hemorrhage of the lenticulostriate artery. Dr. Leon Meyers reported a series of abscesses of the brain in which he showed that those that involve the cerebellar hemispheres show a conjugate deviation of the opposite side, while those of the supratentorial region show a deviation to the same side. I think that is fairly well established. Dr. Shapiro mentioned that conjugate deviation could occur from stimulation of the horizontal canal to the same side, provided the lesion affected only the triangular nucleus. Lesions of the posterior fossa should involve both sides, and if the deviation is always to the opposite side, it would tend to show that you cannot produce a conjugate deviation to one side or to the other by a lesion involving one labyrinth. I am speaking of clinical reactions. There is only one necropsy reported supporting Muskens' experiments, although Muskens, as Dr. Shapiro pointed out, predicted where that lesion would be found.

There is another thing; a number of cases have been reported in which there were postural reactions due to lesions in the red nucleus, and these lesions were reported as quite isolated. I do not know to what extent they involved the globus pallidus or the posterior commissure.

In regard to the case of hyperalgesia acoustica, I wish briefly to report a case that was interesting to me. A man past middle age had suppuration in both ears. In the right ear there was total perforation of the drum membrane. In the left ear was a perforation that was marginal, about 3 mm. in diameter. When I first saw the man he was suffering from a clinical Ménière complex, a total defect of the drum membrane, suppuration on the right and ataxia. He was confined to bed, but there was no rise in temperature, and no spinal puncture was done. He was at home, and we did not move him very much. He has recovered, so that the right ear, which was totally deaf from the Ménière syndrome, which may possibly have been due to a hemorrhage, is dry and has been so for eight years. He is totally deaf in that ear. The other ear with the small perforation in it, from which I had to remove a small crust about once in three weeks, from time to time suffers from hyperalgesia acoustica, although hearing for voice is only 1 meter. He sometimes comes down and has to stuff cotton in his ear because he cannot stand the noise.

Concerning syphilis with trauma, Dr. Shapiro examined that patient also three months before I did. He found the spontaneous nystagmus. I examined with ordinary methods and did not find any spontaneous nystagmus, nor did I find any abnormality with turning tests or caloric tests. That raises the question as to whether we can accept the finding that the nystagmus that appears only when taken with illuminated spectacles indicates definite pathologic changes. I should say that finding spontaneous labyrinthine nystagmus of any degree by the usual methods would indicate a pathologic lesion. That requires observation of a considerable number of cases before one can accept the finding that a nystagmus discovered only by illuminated spectacles, that is, not supported by further findings in testing out the labyrinth, is reliable.

Dr. Shapiro wants me to mention a case we saw together which showed a spontaneous nystagmus with the illuminated spectacles (Licht-Brille), but I am mentioning the one case in which it was not supported by nystagmus found by ordinary methods simply to show that we must not accept that evidence without further experience.

Dr. Sherman Shapiro: The work of Muskens has been corroborated by other observers and is accepted in its essential features by Jacob, who is one of the leading European neuro-anatomists. I wish to repeat that I am not discussing the mechanism of nystagmus that is known to be on a lower level in the brain stem than some of the other vestibular responses. It is a fact that transection of the brain stem does not abolish nystagmus, but it has been shown by Rothfeld that a lesion in the corpus striatum will abolish the quick component of the head movement in rabbits.

Frenzel's illuminated glasses are in use in a number of European clinics and have been favorably commented on in the literature. The case Dr. Lewy cites cannot, I believe, be used as an objection; my examination was made three months prior to Dr. Lewy's, and at that time spontaneous nystagmus in the midline, demonstrated by the glasses, as well as other labyrinthine findings, was present. As many of the symptoms in injuries of the head are on a vasomotor basis, it is entirely possible that all the symptoms had disappeared at the time Dr. Lewy saw the patient. No one would diagnose a lesion of the brain only on the presence of a nystagmus revealed by the use of these glasses. So far, however, I have always noted that in such a case the rotation and caloric and galvanic tests bring out other abnormalities, and I, therefore, believe that they are a valuable help in the study of spontaneous nystagmus.

Saccharomycosis Sensitization. Dr. L. Benno Bernheimer.

This report covers two cases of hyperesthetic rhinitis due to yeast, the symptoms in one case arising as a direct result of the ingestion of pure culture of this organism.

Case 1.—Miss B., aged 25, had had for many years spasmodic attacks of sneezing followed by profuse watery discharge and nasal block. A considerable amount of nasal study and treatment had been given with no relief. Examination of her nose revealed a pale, water-logged, allergic mucous membrane. The left antrum, however, was cloudy, and on washing yielded an amount of gray mucoid material that on culture produced a pure growth of yeast. In order to rule out the element of contamination, the irrigation and culture were repeated two weeks later, with identical findings. A roentgenogram of the antrum taken after the injection of iodized poppy seed oil 40 per cent showed a thickened membranous wall with irregularities, which were interpreted as polypi. A study of sensitization to various inhalents, foods and bacteria gave negative results, with the exception of positive cutaneous reactions to orris root and beef. Beef was immediately eliminated from the diet, and the use of a powder free from orris root was prescribed. The results, however, proved that these findings were merely incidental. Chemical and bacterial filtrates of the yeast recovered from the washings were made, and intracutaneous tests were made with these filtrates. Both pro-

duced positive cutaneous reactions, but the chemical filtrate was most strongly positive and was the one used for desensitization. This attempt at desensitization proved to be futile, although the dosage was increased to an amount that resulted in anaphylactic symptoms.

A decision was reached to operate on the infected sinus, and a sublabial antrotomy was performed. At operation the antrum was found to contain an amount of gray mucoid material, and the lining membrane was thickened and polypoid. This mucoid material, when cultured, produced a pure growth of yeast, as the previous washings had done. The antrum was thoroughly cleaned, and a counter-opening was made into the inferior meatus of the nose. Following an uneventful postoperative recovery, the symptoms of hyperesthetic rhinitis cleared up entirely.

This patient was last seen three months after operation. The nasal mucous membrane had lost its pale, water-logged appearance, and the patient was symptom-free. Although it is early, we believe this to be a permanent result, as her quiescent periods before operation were infrequent and of but a few days' duration.

It is of interest to note that although Miss B. showed a strongly positive cutaneous reaction to the filtrate of this organism, yeast taken by mouth produced no nasal symptoms, while sneezing readily resulted from a local application to the nose of a suspension of yeast in sterile water. The fact that it was not possible to desensitize the patient with injections of the filtrate to which she showed a positive reaction is also interesting. Either this filtrate did not contain all the fractions of the yeast protein to which she was sensitized, or it was not possible to desensitize her by the injection of protein, an experience not uncommon in the treatment of all allergic conditions.

CASE 2.—Miss G. also gave a history of spasmodic attacks of sneezing followed by profuse watery discharge and nasal block. The nasal mucosa was pale and water-logged. The sinuses were normal on inspection, transillumination and roentgen examination. A diagnosis was made of hyperesthetic rhinitis. The patient, however, had had trouble for only three months, prior to which she had had an acne of the face for which she was taking three cakes of Fleishman's yeast each day. On questioning, she recalled that her nasal symptoms had manifested themselves shortly after she had begun taking yeast. Tests of the skin showed a positive reaction to a yeast filtrate and to several fish proteins. The latter finding was discarded, however, as she ate very little fish. She was advised to stop taking yeast, and her hyperesthetic symptoms immediately cleared up. After she had remained free from hyperesthetic symptoms for two weeks, feedings of yeast were again continued in order to establish additional proof of the specificity of this protein. The nasal symptoms promptly recurred within forty-eight hours, and again disappeared when the ingestion of yeast was stopped.

DISCUSSION

DR. MYRON KAHN: As Dr. Bernheimer pointed out, yeast growths have been found in other parts of the body, but it is unusual to find them in the mucous membrane, although they have been reported in the antrum. I saw a patient with a case similar to the one Dr. Bernheimer reported, a girl with hyperesthetic rhinitis. This girl was studying to be a pianist and had great difficulty on account of a constant drip from the nose. On examining the sinus, I did not find anything abnormal. I referred her to Michael Reese Hospital, where she went through all the tests, and we found nothing except sensitization to beef. The sinuses were then studied roentgenographically and found to be cloudy. Then Dr. Bernheimer punctured the antrum and found thick mucus, which, on culture, gave a pure yeast growth. We at first thought it was contamination and repeated the work, when another pure culture was found. We then decided to operate and did a Caldwell-Luc procedure. We found the floor covered with a thick, gelatinous substance. We cultured this and again found pure yeast. This condition cleared

up. I had not seen the patient for some time until today. The nose looked normal; she is free from all swelling, and she says that she does not know she has a nose. She apparently is perfectly well.

The etiology is not known, but I think that these cases are of importance, and I believe that we shall find more yeast of this type if we make cultures in more of these cases and also culture the washings from the nasal secretions.

DR. FRANCIS LEDERER: I should be interested to know what was found on microscopic examination, whether the yeast was found in the cysts and whether this material fulfilled all the properties of a true saccharide.

DR. T. C. GALLOWAY: I should like to know whether Dr. Bernheimer did an exenteration of all the membrane or simply removed the polypi.

DR. L. B. BERNHEIMER: Concerning the micropathology, contrary to the usual allergic membrane, no eosinophilic infiltration was found. The microscopic section resembled an ordinary pyogenic member with infected polyp formation. However, eosinophilic infiltration in hyperesthetic rhinitis is often obscured by secondary infection to which this membrane is so susceptible.

We removed the entire mucous membrane lying in the antrum.

MEDICAL ADVERTISING. DR. AUSTIN A. HAYDEN.

Chapter II, section 4, "Principles of Medical Ethics" of the American Medical Association, definitely prescribes a standard of professional conduct of advertising which, with some few minor adjustments, is not only fair and just but profitable for medical practitioners to use.

Any sort of advertising that does not meet these requirements is unethical for physicians to use and ultimately is unprofitable.

Two outstanding factors make advertising tempting to Chicago physicians at the present time: (1) decreased incomes, a part of the general depression picture, and the feeling that something should be done to stimulate better medical finances —no one seems to know just what—and for that reason everyone is willing to try almost anything; (2) Public Health Institute notoriety; apparently the advertising brought hundreds of thousands of patients. Why will it not do the same for the medical profession in general?

Why is direct advertising contrary to the code of ethics? For these reasons, principally:

1. To secure equal opportunity for all medical practitioners. Their education and industry and not the amount of money they could spend in advertising is admittedly a more just basis for building a medical "business" than direct advertising, where money alone would count. The affluent physician would be at a great advantage.

2. If advertising became ethical, every physician would be compelled to compete with his neighbor. In the end, only those who sold the advertising would benefit, and the costs necessarily would be passed on to the ultimate consumer, the patient.

Slides were shown illustrating various types of vicious newspaper publicity. As the ads became less glaring and more conservative, their effectiveness decreased. The medical advertising board of the Public Health Institute has done real service to Chicago in this respect—to both the public and the medical profession.

The immense amount of real news that the medical profession has and that the newspapers and magazines are extremely anxious to get is illustrated splendidly twice yearly by the information emanating from the American Medical Association at the national meeting in the summer and the educational meeting in midwinter. This continues through the year in less amounts. Morris Fishbein and the Board of Trustees are responsible for the slides shown of advertising by Parke, Davis and Company, Squibbs, Mead Johnson and Company, Eastman Kodak and Metropolitan Life Insurance, bringing health messages to the public that should indirectly greatly benefit physicians; others will follow.

Even though the times are difficult and some physicians are sorely tempted to take advantage of their fellow practitioners (some few actually have), the majority will fare best by strict observations of the code of ethics of the American Medical Association, and direct advertising will not be found to be a cure for our present troubles—"the most effective advertising possible is the establishment of a well-merited reputation for professional ability and fidelity."

DISCUSSION

DR. HERBERT E. PHILIPS (by invitation): Dr. Hayden invited me to listen to his paper, but the invitation to discuss it was entirely unexpected on my part.

I am glad to have listened to the discussion as to what constitutes health education propaganda that can be endorsed by professional men, and what is display advertising that lessens the dignity of professional men and should not be endorsed. At present in the American Dental Association we are having a sharp discussion over what kind of propaganda is proper to use. I do not know whether any of you are acquainted with the kind of material used by dentists in Little Rock, Ark., but there they use what is called dental health advertising in the newspapers and is paid for by the dentists. It was taken up by our American Dental Association and was recommended to various dental organizations throughout the country. There has been a good deal of criticism of the material because many felt it was display advertising and not the dignified type that Dr. Hayden illustrated this evening. I feel sure that as a result of the present depression, with the loss of money by the large group that has been able to pay fees to both physicians and dentists, we shall likely be urged to turn more and more to advertising, and I feel that the work Dr. Hayden is doing in pointing out what is proper health propaganda and what is display advertising throws the type of material that can be used into a clear light, and also throws into a clear light the type of material that should not be used.

DR. G. HENRY MUNDT: I am tremendously interested in this subject. There has been a great change in the attitude in medicine in the last few years. I remember in 1926 I started to travel around in this state and all over the middle west talking of medical economics. A good many men told me they thought I was on the wrong track, that the thing was nonessential and that none of the better groups of medicine would ever consider it, but all one needs to do now is to look at the activities of local medical societies and national organizations all over the country to realize that there is a marked interest in the thing. Certainly what Dr. Hayden said about the code of ethics being the basis on which a man should stand to advocate the advertising is well taken. I believe that everyone who has seriously thought on the subject has felt that if you measure any advertising by that section of the code of medical ethics it can stand on its own feet against, it would be a fair type of publicity. I want you to get the idea that I am not advocating any type of newspaper or display advertising. An effort was made in one part of this country and a considerable amount of money was appropriated to try placing good copy in newspapers all over the state, but after a year or two this plan was found to be of little value, and it was discontinued. I wish you to know that if it had not been for the prominence of the names of the men interested in the Public Health Institute they could never have gotten to first base, and would never have gotten their advertising matter in the papers. There was another thing, and that was that the Public Health Institute was doing Wassermann tests for large numbers of physicians in this town. There is no question about that. I think that Dr. Hayden misspoke when he said that Dr. Kretchmer straightened out the Lewis Institution. It was Dr. Kreuscher who did that. I know there was great furore all over the town relative to the advertising of the Lewis Institution, and there was a feeling that the men whose names were listed there should be excluded from organized medicine, but they were absolutely innocent.

Within the last two years the American Medical Association, which stood firmly on the basis that they could not consider economics because it was not included in the constitution, has appointed a Bureau of Medical Economics. It is my impression that the material that is gathered by that Bureau on Medical Economics and correlated by various groups all over the country will get some place. But I wish to say this to you, the dentists have us backed off the boards. Dr. Phillips is chairman of a committee from the American Dental Association, and they have spent a good many thousands of dollars in the study of economics in relation to the profession. That is what we must do. Somebody has said that we were sitting on a firecracker, but did not know it. I believe there is some truth in that. I believe that the rank and file of the men in the medical specialties little appreciate how definitely they are faced with a change in medical practice.

Dr. H. B. Young, Burlington, Iowa (by invitation): I have been seventy years in touch with medical practice, sixty years on my own and ten years as the follower of my father, who went to Monmouth, Ill., in 1839, to practice medicine. I have thus been an eye witness to much of the evolution in medicine, and I still hold to the doctrine he laid down, namely; Make yourself a better doctor every tomorrow than the today, be unconcerned about your colleagues' patients, and never offer knowledge or service save in the presence of widespread disaster, when common humanity may demand both. This is my fifty-third year of practice in Burlington, and I think that if you would peruse the files of the papers there you would find my name very infrequently.

In connection with the state-wide publicity demonstration Dr. Mundt spoke of, I think I know why it brought no appreciable results. The language of medical publicity is not understood by the public at large. It understands that used by charlatans and merchants, and anything less direct is talk outside of its level. There are thus just two roads from which to choose; either use no publicity, or beat the other fellows in straight advertising—with this one restriction: whatever you advertise is proper so long as it tells the truth.

I might add that since July 1, 1931, in Iowa, owing to the wisdom and energy of the legislative committee of the state society, it is unlawful to sign my name or have a card, letter head or sign for professional purpose without having "M.D." attached. We must now have distinguishing marks to avoid being mistaken for one or another of the other kinds of "doctors."

Dr. Francis Lederer: It evidently took the depression to get a rise out of the medical profession, but I remember we were interested in the problem several years ago and went to the telephone directories and newspapers to investigate. It was interesting to see how many nose and throat men were advertising in Chicago newspapers, and also the number of articles and stories by so-called plastic surgeons that appeared, telling "How I was made young in twenty minutes," the story of a girl who went to a doctor's office and without any pain or bandaging came out twenty years younger than when she went in. I think we never did anything about it except to clip the advertisements. I believe that the thing that has roused us to the point where we make lantern slides out of the advertisements has been the slow loss of business and the high cost of advertising. They have brought about their own downfall, or else the economic situation has.

I should like to ask about the little cards that appear in foreign newspapers. I am aware of certain advertising in Bohemian newspapers in this town, with cards that carry the names of men and their specialties. I should like to know whether that is considered ethical? Moreover, I think that the Chicago Medical Society is guilty of violating certain ideals in that it would admit an advertisement of a man who has been guilty in the past of some unscrupulous practice and now is made ethical by a card appearing in the *Bulletin* of the Chicago Medical Society, stating to what he limits his practice. A good deal of talk is going around about the electrical treatment of tonsils, which is being advertised in various ways. I think that we are being aroused mainly by the fact that we are realizing all these things at a time when practice is at a low ebb; and I do not think that the ads of

Parke, Davis and Company or of Mead Johnson and Company are solving our problem. As Dr. Young said, we should either do no advertising or give the laity truthful signed statements in the papers.

DR. SAMUEL J. PEARLMAN: We have just seen the interesting slides of Dr. Hayden, showing just what he considers good advertising from the standpoint of the medical profession and what he considers bad advertising. Most of us, I am sure, are agreed that there is only one way in which a physician may advertise himself, and that is by the time-honored and ancient method of doing the best work he is capable of and in so doing gaining the esteem of his patients, their families and friends as well as that of his colleagues. There seems to be no way of using advertising in the commercial sense that is not open to the most serious criticism. About all that is sure about this type of advertising is that various newspapers and journals will profit. Even the advertising undertaken by certain well known firms has an ulterior motive, and much as it may extol the virtues of physicians in general, its primary purpose is to gain good will for the firm paying for the publicity. I gathered in a distant sort of way that Dr. Hayden intimates that we may be due for startling changes in the near future, owing to present economic stress, but the samples he showed us of advertising by clinics and individual physicians seemed all to be in the objectionable class. Until some new plan as yet unformlated appears, it is obvious that physicians, clinics, hospitals and the medical profession in general must continue to seek good will and respect from the public by conducting themselves in such a fashion in their daily life so as to merit these.

DR. WALTER H. THEOBALD: May I ask Dr. Phillips to tell us what the Dental Association thinks of the Pepsodent program given on the air—"See your dentist twice a year."

DR. HERBERT E. PHILLIPS: I think that depends on the person you ask. The profession has taken no position on it. I personally do not endorse it.

DR. AUSTIN A. HAYDEN: In regard to what Dr. Pearlman asked about, what constitutes bad and good copy, that is a pretty difficult thing to answer in just a word. I should say that the underlying principle is that when a man graduates from medical school he has his diploma and the knowledge he got in medical school. The proper rule for him to follow in advertising is to treat the other fellow the way he would like to be treated himself. That is, if you are particularly successful or have access to money, you should not buy space and laud your own ability, but should compete with your fellow practitioner on the terms of service to your patients rather than by paying for advertising. Why is the Metropolitan copy good and the Wicker Park bad? The Metropolitan is impersonal advertising so far as the physicians are concerned. Whether it is good for the Metropolitan Life Insurance Company I do not know, but I assume that it is. One reason why the Metropolitan is endeavoring to engratiate itself in the medical profession, I think, is that before long a sickness insurance school will be launched in this country on a large basis, and perhaps the large insurance companies, among which is the Metropolitan, will be underwriting this school, so the good-will of the profession is of value to them. It is said that the company has spent $4,000,000 in the last several years on such advertising as this. That is a tremendous sum, but it is not a tremendous sum when divided among twenty-five million policy holders, which it is said to have.

As to the electrical treatment of tonsils and the ads of Parke, Davis and Company not solving the trouble, which Dr. Lederer mentioned, let me speak of the electrical treatment of tonsils first. I am aware that there are a number of suburban and small town newspapers that are carrying such advertisements as he mentioned. These are strictly unethical. In the first place, they are not truthful. In the second place, they certainly take advantage of the man who does not advertise. You know that the medical profession is sitting pretty so far as advertising is concerned. It is true that if Dr. Harry Pollock advertised in the *Tribune* that he cures or corrects nasal deformities he would have a lot of noses

to correct, so long as the rest of us did not advertise the same thing. If we did we should all be starting from scratch and one would be no better off than the other. The only winner would be the newspaper or the medium through which the advertisements were carried.

I wish I might think as Dr. Young does about an absence of an impending catastrophe. I think that great changes are about to come on the profession and on the public in the practice of medicine. Dr. Phillips told us that the League of Nations is absolutely committed to a sickness insurance. All of this must affect medical practice, and all of us as practitioners of medicine. I think that we had not paid enough attention to medical economics. Do not misunderstand me that I am advocating state medicine; I should be the last to see the medical profession robotized, but I think that we should keep in touch with economic conditions. We have many ways in which we can advertise impersonally. It is quite conceivable to me that if the type of advertising used by the Public Health Institute were open to all departments of medicine, the Chicago Laryngological Society would have been forced to put on a campaign of publicity in defense of individual practice in the things happening to the genito-urinary fraternity here in town.

The way I think we can advertise is by good, conscientious work and a willingness to serve the public and serve them well at a price they can afford to pay. I think that is the best advertising any physician or any business can have. I believe, in the second place, that active membership in medical societies goes a long way in advertising the physician in the way he should be advertised, by producing respect in the minds of fellow physicians. We cannot fight each other. We must get along among ourselves. Let me add that the capitalization of those things should always be in the public interest, not for our own personal advancement financially. If we use that as the direct end, we shall fail I believe. We must always be concerned in the welfare of the public, and if we do well by them we shall fare well ourselves. The public contacts that have been opened to us are tremendous. They include lectures and various other types of public appearance over the radio, which I think we do not sufficiently avail ourselves of. The Illinois Medical Society has had a great deal of difficulty in securing physicians, particularly to lecture on eye, ear, nose and throat conditions. Some of us have done it, but the thing has been neglected.

In a word, I believe that the basis of good copy and of sound medical advertising is the basis of merit and work. I think that physicians should be known, not by the dollars they are able to spend in direct advertising, but rather by the fruits they bear.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

H. P. Schenck, M.D., *Reporter*

March 16, 1932

Ralph Butler, M.D., *Chairman*

Intimate Notes on the Ear, Nose and Throat Clinic. Dr. William Hewson.

These comments are a plea to consider the ear, nose and throat dispensary more seriously. There are some in the medical profession who might be called dispensary nihilists. They would abolish all dispensaries as unnecessary and detrimental to the best interests of the medical profession and the laity. They

base their contention on three fairly well recognized observations: (1) the growing abuse of the free clinic; (2) lack of cooperation between the hospital and the outside physician and (3) the comparative small number of cases referred to the house from the dispensary.

Abuse of the dispensary has existed ever since it was organized, and most of us will admit that today more people who are able to pay are using the free clinic than ever before in its history. This number is increasing every year, and very little, if anything, is done about it. It seems to be a social problem and, as such, is referred to social service departments and agencies. I believe that the average social service department of a hospital is incapable of successfully dealing with the matter, because even if there were enough workers and enough time to ask all questions, the patients would continue to falsify their answers in order to get free treatment.

Lack of cooperation between the hospital and the outside physician has hampered the progress of both for many years. The outside physician wonders why a patient sent to the hospital for one condition is retained by the dispensary for another. He, of course, chafes at the discovery of several of his pay patients being treated in the dispensary free. He feels ignored by the hospital that takes his patients, sends him no word about them, perhaps operates on them without his knowledge and in the after-care gives him no hint of why the hospital wants them in the dispensary. He perhaps resents most of all the competitive spirit of the dispensary, and he cannot reconcile a charitable institution underbidding him.

Cases referred to the house from the dispensary are quite few if tonsillar conditions are not included. This is particularly apparent in hospitals in which tonsillar conditions are dealt with by the receiving ward only. Chronic otic and sinus conditions make up the next largest group. Patients with these conditions are benefited by treatment in the dispensary, but most reliance is put on directions for care in the home. Visits to the dispensary cannot be arranged often enough for the patients, because the days of the dispensary are too few or too crowded to be effective. It is contended that most patients with acute cases should not be ambulant and therefore should not be treated in the dispensary. The dispensary nihilist finally calls attention to many of the western states, in which the large outpatient clinic is the exception rather than the rule.

In the course of time since the organization of the dispensary, one has to admit that the object of the dispensary, as well as its public acceptance, has broadened. Patients no longer dread the idea of going to the hospital, if they do not have to stay there. In fact, the dispensary idea has been successfully sold to them, "oversubscribed" one might say, and sold cheaply. The average dispensary is overcrowded with long lines of waiting patients. Patients cough and sneeze; children run about and get in the way; weak patients, requiring only postoperative instructions, stand about. Patients with contagious diseases, those with chronic running otic condition and those with foul-smelling atrophic rhinitis are all gathered together waiting their turn. Inside the consultation room there is almost as much of a crowd, and also much confusion. The various activities being carried out with each patient, usually in view of others, need not be detailed. It is worth recording, however, that the physicians in the dispensary are more than busy and are working under pressure.

Some wit has described the dispensary as a place where patients are dispensed with and gotten rid of in the speediest time possible. Such a dispensary may be spoken of as "going to seed." The most important sign of such a condition is lack of interest on the part of the physician in the dispensary. They are perhaps overworked; the equipment has not been kept up, and contacts and encouragment from other members of the staff are wanting. It becomes easier for them to get in late and leave early. Records are poorly kept, or no notes are made. The incentive for work is lost, and the morale of the dispensary suffers. Tired or cross physicians do not reflect credit on themselves or the institution. It makes little difference how important a dispensary may be, it will lose its worthwhileness if the morale is lowered, the work becomes drudgery or the general atmosphere

becomes electric and combative. It is useless to try to interest younger men in such a dispensary, no matter how many and varied are the opportunities. Their interest will be only short-lived, and one cannot blame them. In such an institution the hospital authorities are beside themselves to know what to do. The house chiefs are usually ignorant of what the situation in the dispensary really is, and they would rather not think about it. It is quite evidently nobody's business, so that the dispensary soon becomes an overcrowded torture chamber to the patient, a sweat shop to the physician, a sore thumb to the hospital and a neglected step-child to the staff of the hospital.

The object of the up-to-date dispensary has been extended to include not only a philanthropic effort for the poor patient, but also a broadening educational opportunity for the physician. No physician in a dispensary works for nothing if he is truly interested in his work and has the opportunity and capacity for doing it well. A suggested plan for a moderate-sized clinic would be, briefly, as follows: The patients would be divided into two groups: those who could visit a free clinic and those who could visit a pay clinic. Each clinic would meet twice a week, Monday and Thursday or Tuesday and Friday. Both would be presided over by the same staff under the direction of the chief of the clinic. The free clinic would be an examination and advice clinic where few, if any, treatments would be given. Physicians working on a problem of treatment could, of course, have patients admitted for this purpose, but the number of such problems would be limited by the chief of the dispensary. Myringotomy and washing of the antrums, as well as single treatments in acute cases, would, of course, be done. An appointment system managed by the staff of the hospital would limit the number of cases to the capacity of the clinic and make it possible for the patients of both pay and the free clinic to know within fifteen minutes of the time they could be seen, so avoiding the long waiting and crowding. Patients entering the pay clinic would be asked a fee comparable to the office charge of the outside physician, about fifty cents. The dispensary physician, with some appropriate title, would be on a salary paid by the hospital. His appointment for service would be for six months, and he would be allowed appointments in only one other hospital. A system for checking time would be adopted, so that the physician and the hospital would both be protected. Dispensary assistants without title would be solicited from among outside physicians, physicians on other staffs in the hospital, resident physicians and others wanting to observe a number of cases of otic or sinus conditions, etc. These would be under the direction of the chief of the clinic, but would be assigned to the dispensary physicians for instruction. Dispensary assistants would not be limited and would not be admitted to the pay clinic. Monthly meetings would be held either separately or in conjunction with the meetings of the hospital staff, and one report would be required from each dispensary physician during his term of service. Volunteer lay workers would be assigned to do secretarial work and to make certain hearing and statistical charts.

The hospital would furnish well lighted and well ventilated rooms for consultation and treatment. These would be divided by movable screens or curtains into treatment cubicles. There would be a sufficient number of cubicles for each dispensary physician to have two. A dark room and a hearing room would be conveniently located. The usual instruments would be adequate. Provision would be made to examine all children under 6 years of age lying down.

DISCUSSION

DR. JAMES A. BABBITT: Some of the routine details mentioned in Dr. Hewson's paper may be at variance with our old ideas, but the practical points are well worth considering. It is pleasant to sit down with plenty of time and work on a dispensary case without competitive, social and other factors. I like the term "clinic" and am not certain that it does not properly apply to the outpatient department. Dispensaries may be the most important part of a hospital. Well appointed facilities with close cooperation with related dispensaries are essential.

DR. PHILIP STOUT: The hardest problem is the pay clinic. There is no reason why every poor person should be taken to a hospital when the city government is paying physicians to do this work. The pay patient at once wants more than a student; he wants the professor, even if he is paying but fifty cents. Physicians on the outside are getting a trifle tired of having hospitals take their patients. It is difficult to separate at the door those who are able to pay and those who are not able to pay. Separate the goats from the sheep; treat the sheep and send the goats home.

DR. MATTHEW S. ERSNER: Dr. Hewson has brought an enticing utopia before us. The drudgery of the physicians, the poor waiting patients, the crying children and the other features mentioned combine to make a gloomy picture. We all know that conditions are not satisfactory. If people are taking to free treatment, the tendency is toward state medicine. If we think that progress is being made toward state medicine, then the medical profession should fortify itself so that physicians may dictate certain terms. I have put in ten or twelve years in dispensary work, but find that we hurry in our offices as well as in the dispensaries. Perhaps we should socialize medicine.

DR. WILLIAM HEWSON: The problem of the dispensary is one that we are not meeting adequately. When Dr. Ersner says we are drifting toward state medicine, he is right. We cannot antagonize outside physicians. The clinic must be thought of differently than formerly. The hospital has a problem to solve in its relation to outside physicians. Dr. Babbitt mentioned the pleasure obtained in taking time for patients in the dispensary and for thoroughly studying the case. If men working in clinics could spend time over individual cases and have apparatus adequate for study, there would never be a paucity of dispensary physicians. In reference to Dr. Stout's comments, there is no doubt that the physician in present circumstances is competing with himself.

ACTINOMYCOSIS OF THE TONGUE AND TONSIL. DR. ROBERT F. RIDPATH.

After a review of the literature on actinomycosis, the case of a colored man, aged 42, was reported. The patient, when referred from the dispensary to the ward, was unable to talk and was admitted with the diagnosis of actinomycosis of the tongue and left tonsil. Eight days before admission there had been severe pain in the tongue and the left side of the face which was attributed to an injury by a new tooth brush two weeks previously. Physical examination gave negative results except for alterations in the mouth, neck, pharynx and tongue. The tongue was greatly swollen and completely filled the mouth. The left tonsil was enlarged, engorged and covered with mucus and ulcerations. Large, hard masses were noted along the left border of the tongue. The posterior cervical glands were palpable. The supraclavicular and infraclavicular fossae were filled. Tracheotomy was performed because of the dyspnea, but the swelling continued. Eleven days after admission there were softening and contraction of the area along the border of the tongue. A spinal puncture was made and clear fluid obtained under a pressure of 12 mm. The Wassermann reaction of the spinal fluid was negative. The urine was negative for iodine, although iodine was given in large doses. The typical ray fungus was isolated, and granules were identified. The temperature was never over 101 F. The total white cell count was 16,000 on admission, with 84 per cent polymorphonuclears, 9 per cent small lymphocytes and 7 per cent large lymphocytes. A week later the white count was 8,400, with 53 per cent polymorphonuclears, 32 per cent small lymphocytes and 32 per cent large lymphocytes.

The patient received a liquid diet and a phenol mouth wash. Incision and drainage of the peritonsillar abscess were followed by tracheotomy. Sodium iodide was administered intravenously in doses of 30 grains (1.95 Gm.) three times a day. Twenty days after admission, the tracheotomy tube was removed, and ten days later the patient was discharged from the hospital. The tongue was still thick and hard, but there were no new lesions and the general health was good.

DISCUSSION

DR. JOHN A. KOLMER: I have deep interest in actinomycosis of the tongue. The two cases of actinomycosis of the jaw that I have seen were both in farmers. This is the first case of lesions of the tongue that I have heard reported. Serum therapy, vaccine therapy and chemotherapy are of no avail. Potassium iodide is the important agent. One patient received as much as 500 grains (32.5 Gm.) a day. Heroic doses are necessary. I have no explanation for the surprising negative result of urinalysis for iodine under such dosage.

THE PROPHYLAXIS AND TREATMENT OF SEPTIC MENINGITIS. DR. JOHN A. KOLMER and DR. K. AMANO (by invitation).

The mortality of streptococcic and pneumococcic meningitis has stimulated the search for adequate therapy. A clinical and laboratory experience extending over a period of ten years is here summarized. The majority of cases of streptococcic and pneumococcic meningitis are secondary to otitis media or sinusitis. The organisms may enter the postnasal area and then the meninges. Their invasion by any route presents the most important surgical problems. When there is impossibility of extirpating the focus, reinfection of the meninges from these areas occurs. Prevention has been thought possible by the administration of immune serums, given before operation. The signs and symptoms of meningitis may develop during pneumonia, but the majority of these prove to be meningismus. The majority of recoveries reported in the literature are of this type. Streptococcic meningitis may develop during scarlet fever without lesions of the ears or throat. The majority, however, are secondary to primary infection of the ear, mastoid or sinuses. Surgical drainage must be established. I have never seen a staphylococcic meningitis that was not secondary, and these infections tend to remain localized. The signs and symptoms may be indicative of localized meningitis, but the spinal fluid may be clear early and mislead diagnosis so that meningismus is suspected. It is difficult to decide on a radical form of treatment unless the signs and symptoms of diffuse meningitis are noted. It is impossible to save any animal if more than from twenty-four to forty-eight hours have elapsed after the onset. We have never seen any harm result from the withdrawal of 5 cc. of spinal fluid. There has been no dissemination of infection. The pressure is measured with a mercury manometer. In septicemia no infection of spinal fluid results from lumbar puncture.

In early localized meningeal congestion, pressure may or may not be increased, and the spinal fluid may be clear. In localized meningitis, the fluid obtained a few hours after the onset shows few cells, increased protein and sometimes an increase in sugar. Absence of organisms in direct smears may be all that is lacking, but should not be permitted to give a sense of false security. The danger lies in waiting. Infection continues to spread during the remaining few days. Radical surgical procedures and serum therapy should not be delayed. In specific chemotherapy there is no one specific substance. Reliance must be be placed on surgical drainage and serum therapy. There should be complete extirpation of cells if the mastoid is involved, and the best possible drainage when sinusitis exists. Wherever exposure of the dura is possible, wide excision for free drainage of spinal fluid should be made. There is a difference of opinion concerning the advisability of lowering the spinal fluid pressure. Experiments of our own are not conclusive, but it is probably feasible to obtain results by draining in this fashion. If infection is progressive, more radical procedures are necessary. Spinal drainage for from six to eight hours with an increase of fluids or laminectomy with forced fluids is indicated. We may even consider continuous spinal drainage. In our dogs, immune serums alone failed. Serums plus drainage also failed. Stewart's method of using serum with ethylhydrocupreine when drainage was also employed was found excellent, and there were recoveries amounting to a high percentage. Type I pneumococcus in the experimental dogs was fatal in from four to seven days, while the dogs used as controls died in from twenty-four to forty-eight hours. Under ether anesthesia a double frontal trephine was made, and the needles were introduced

into each cerebral subarachnoid space. This was repeated in twenty-four hours. Many of the dogs recovered permanently. Some animals treated similarly with physiologic solution of sodium chloride alone recovered. The treatment of human beings was not as effective, but all five patients had late cases and were practically moribund. The subarachnoid space was obliterated by plastic exudates. In dogs there was no doubt of success, because the treatment was instituted before the plastic exudates formed.

In pneumococcic and streptococcic meningitis, serums and chemical agents may be introduced into both common carotids with surprising ease and safety. There is no bleeding when the technic is mastered. Given this way, there is no great toxicity of the drug, and bilateral injection, as well as cisternal drainage, may be used.

In dogs Felton's combined antipneumococcic serum has shown definite value, while the antistreptococcic serums have not been effective. The Parke, Davis antistreptococcic serum has given somewhat better results than others. Of course, in these dogs there was no primary focus in the mastoid cells or sinuses, and therefore the condition did not parallel the cases in human beings. Of ten dogs with streptococcic meningitis, two received Pregl's solution and concentrated antistreptococcic serum by intracarotid injection. Injections of neutral acriflavine may be used with greater confidence.

In the prevention of streptococcic and pneumococcic meningitis bacterial examinations should be made both before and after operation. Specific prophylactic measures must be instituted early. Intravenous and intramuscular injections of Felton's polyvalent serum influenced the incidence of meningitis in rabbits and dogs. We should take a culture from sinus before operation and culture the material in a mastoid wound at operation. The oral administration of pneumococcic vaccines increases the resistance of animals to pneumococci and may raise the immunity higher than when given subcutaneously. Similar results are not obtained by the oral administration of streptococcic vaccines.

The clinical applications of this work may be summarized as follows: 1. Vaccination should be carried out. Operation may be considered one week after the last dose of vaccine. In pneumococcic infections it may be better to give the vaccines orally. 2. Serum prophylaxis should be used. 3. Type the pneumococci in such infections. 4. In type I pneumococcus infection give 10 cc. of Felton's polyvalent serum and repeat the dose on the second and fourth days. In types II and III pneumococcic infections the serum should be used in doses of 15 cc. The same doses may be used in children over 15 years of age. 5. Ethylhydrocupreine may be given in addition to serum. This is best given in 5 grain (0.32 Gm.) doses in milk every five hours after operation.

DISCUSSION

DR. PHILIP STOUT: There are two schools as far as this question is concerned; one waits; the other goes ahead. It is still questionable which is correct.

DR. MATTHEW S. ERSNER: Dr. Kolmer has brought us a valuable message. We had two cases at Mount Sinai Hospital that may be of interest. One patient had meningitis following mastoiditis, with 1,800 cells in the spinal fluid. The next day, 30,000 cells were noted in the spinal fluid, so the carotid was opened and polyvalent serum and acriflavine were introduced. The second case occurred four months ago, and we are still waiting for secondary abscesses of the brain to form. It might be well to bear in mind a few precautions. All of these patients, whether they have meningismus or cerebral hydrorrhea, have increased intracranial tension and therefore should receive dextrose, magnesium sulphate and plenty of spinal punctures. This treatment should be followed up with polyvalent serum. I do not believe that the question of the sinus can be emphasized too much. One case that started as pansinusitis developed into meningitis. When the sphenoid was opened, sphenoiditis was found. These conditions are best treated with much shrinking, but no surgical procedures. In one patient we found difficulty

in regard to the intake of fluid. As soon as the intake was over 500 cc., severe headaches occurred, and the visual fields became contracted.

I am interested in the possibility of administering vaccines by mouth. In 1916, Coates and I read a paper describing various methods of administering vaccines, but our results were not gratifying, and I hope that these newer methods will prove satisfactory.

DR. WILLIAM HEWSON: The question of whether we are dealing with a case of meningismus or one of meningitis is always a bugbear. A boy, aged 6 years, at the Abington Hospital, gave a history of ethmoiditis on the right side for which he was treated in the hospital. He was sent home, but returned later in convulsions. The ethmoidal area was exenterated at once, and intranasal maxillary drainage was performed. The meningeal irritation continued for four days. Later it was learned that otitis media had occurred four months previously. The drum at this time was doughy, and although it was incised, nothing was recovered. Four days after paracentesis a discharge began to flow from the ears, and mastoiditis developed. Type III pneumococci were isolated from the wound. The spinal fluid showed a pressure of 40 mm. and 400 cells. The patient was normal after an operation on the mastoid.

DR. SAMUEL R. SKILLERN: Pneumococcus is the organism that has a preference for bone. We are glad to know how to govern meningitis, because now we can govern osteomyelitis. I have frequently called Dr. Kolmer in on these cases. In a child with plastic exudate seen a short time ago, we were too late in operating. How shall we know when to operate and when not to operate? We frequently see a postoperative rise in temperature. If we wait forty-eight hours, we may be too late.

Directory of Otolaryngologic Societies *

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. Walter G. Howarth, 21 Devonshire Place, W., London.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. Norman Patterson, 82 Portland Pl., W. 1, London.
Secretary: Dr. T. B. Jobson, Netherwood, Epsom Road, Guildford.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: Sept. 19-23, 1932.

American Bronchoscopic Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.

American Laryngological Association

President: Dr. George E. Shambaugh, 122 S. Michigan Ave., Chicago.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Edmund Prince Fowler, 140 E. Fifty-Fourth St., New York.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.

American Otological Society

President: Dr. L. W. Dean, Washington University, St. Louis.
Vice President: George L. Tobey, 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 Fifth Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: Seattle: Time: June 30-July 2, 1932.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. J. Peirce, 511 N. Main St., Pueblo.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. R. T. Atkins, 4 W. 53d St., New York.
Secretary: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. H. L. Harley, 101 S. Indiana Ave., Atlantic City.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Fort Worth. Time: December, 1932.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, 3117 West Ave., Newport News.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Howard C. Ballenger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.
President: Dr. Edward D. King, 707 Race St., Cincinnati.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. Ivor G. Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. Raymond Goux, 1551 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. Charles Imperatori, 17 E. 38th St., New York.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. W. P. Haney, 107 S. 17th St., Omaha.
Vice President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott Applewhite, 706 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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